Subscription Price:
United States and Canada, $5.00 Per Year.

Great Britain, 21 Shillings; Single Copies 3/6.
Foreign, $6.00 Per Year; Single Copies, 75 Cents.

VOLUME I JULY, 1919 NUMBER 3

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THE JOURNAL OF INDUSTRIAL HYGIENE

AND ABSTRACT OF THE LITERATURE

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Application for entry as second-class matter at the postoffice at New York pending. Published monthly.
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The JOURNAL *of* INDUSTRIAL HYGIENE

PUBLISHED MONTHLY

VOLUME I JULY, 1919 NUMBER 3

THE PROBLEM OF ASCERTAINING THE ACTUAL RISE IN MORTALITY CAUSED BY UNHEALTHY TRADES *

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IN the study of industrial health hazards, probably the most difficult problem that presents itself is to arrive at some conclusion as to the actual effect on mortality caused by the handling of industrial poisons, and by working at trades that are burdened with an occupational disease.

To the purely scientific investigator this question has not the paramount importance that it possesses for the practical man who must deal with these figures in the earning of his bread and butter. To the scientist the occurrence of one case of industrial disease is sufficient reason for the institution of vigorous remedial measures, remedial legislation, if necessary, and profound research. This is very properly so, as the ideal condition for which we are all striving guarantees every operative perfect safety from both accident and health hazards in the performance of his work.

The practical man's problem, however, is not of the future but of the present. Take the life insurance underwriter, for example. The occurrence of one or two cases of industrial disease, or one or two deaths from industrial disease is not sufficient reason for him to deny to all the operatives in the plant the benefit of insurance. He must estimate the actual ravages of the disease and charge a rate therefor. In many instances this is still largely a matter of skilled guessing. One of the most prominent examples of a disease which illustrates this difference in viewpoint is anthrax. An enormous amount of research work has been done and a great deal of money has been spent on this disease, but from the viewpoint of the practical man it can be ignored with comparative safety. The cases are so few compared with the number of those who are exposed to it, that its effect on mortality is practically nil. In the Medico-Actuarial Mortality Investigation published in 1913, the class of journeymen tanners returned a mortality materially below the standard and, although the class was small and liable to fluctuation, there is no indication of high mortality from any inherent cause. Experience of individual companies also bears out this conclusion.

There are many reasons for this difficulty in estimating the actual ravages of an industrial disease, all exceedingly hard to overcome. First, there is the problem of labor turnover. It has been demonstrated times without number that working conditions bear a distinct relation to labor turnover. In the factories, therefore, that maintain the most primitive systems and where the problem might be studied to best advantage, the huge labor turnover makes any investigation impossible. It frequently happens that the worst factories will show the fewest cases of industrial disease for the reason that their operatives do not remain long enough to contract it. It is useless to quote figures illustrative of this condition as it has been shown conclusively to exist. It

*Received for publication April 23, 1919.

may be stated, however, that during the war, while labor was particularly unstable, the condition was especially noticeable, monthly overturns of 50 per cent. being frequently noted and, in certain plants, going even higher. This condition is a very unfortunate one for the industry from the standpoint of efficiency, but fortunate in that it minimizes the effects of poisons which would otherwise have a free rein in working disastrous results.

The second great cause of difficulty in estimating the ravages of industrial disease is the fact that, in probably a large majority of instances, although an operative may have actually contracted an industrial disease, he dies of some acute malady to which in his weakened condition he is an easy prey. There are industrial processes which may cause disease of practically all the vital organs—lungs, heart, kidneys, digestive apparatus—leading to a weakening of these organs which materially aids the acute plagues, such as pneumonia and typhoid fever, in increasing the number of their victims. This phase of the problem is further complicated by the fact that the victim is frequently no longer employed in the industry which, in reality, caused his death. Many operatives, especially the more unskilled, leave an occupation when they find it adversely affecting their healths.

Remedies have been suggested for these difficulties. A standard state death certificate, giving not only present but previous occupations during a period of several years, is a frequent suggestion. The American Association for Labor Legislation has done some missionary work in this connection. The data available, were such a certificate universally adopted, would undoubtedly be of the greatest value. Sixteen states also compel the reporting of industrial diseases, and as these states cover the largest industrial centres an immense amount of invaluable data is constantly accumulating. This data will show the prevalence of diseases peculiar to certain industries and if properly matched up with the vital statistics will assist materially in the final solution of the problem.

One would suppose that one of the most valuable sources of information bearing on the problem would be the experience of the great life insurance companies themselves. This is true in great measure, but forces are at work which tend toward inaccuracy in these as in other figures. In the first place, insurance companies have to hark back so far into the past to get sufficient exposures on which to base their findings that they do not properly reflect the trend of conditions in the industry. As a practical example of this, any figures giving mortality in the felt hat industry which go back twenty years would not be of great practical value today. Twenty years ago in all felt hat making centres mercurial poisoning was the mightiest of menaces to health and the offices of local physicians were flooded with operatives needing treatment for the condition. Today, with modern appliances for confining and removing the fine mercurialized dust, the dangers have materially abated and in the very best equipped and best managed factories are almost non-existent. The same is true of many other industries.

Of course the error producing factors of labor turnover, instability, etc., are present in the life insurance experience as well as in all other statistics. Furthermore it must be remembered that, in the past, operatives in the hazardous and unhealthy trades have been denied insurance in a number of the largest insurance companies. This is especially true of the old line companies. This state of affairs is rapidly correcting itself as it is becoming more and more the modern life insurance opinion that its benefits should be extended to everyone as fast as the rate necessary to be charged can be ascertained. The industrial companies are in a position to furnish probably the most valuable data owing to the immense number of lives which they insure, and many valuable studies have been contributed to the subject by Dr. Hoffman of the Prudential Life Insurance Co., of Newark, N. J., and by the Metropolitan Life Insurance Co. The experience which will be available with the development of group insurance seems to aim more directly at the problem than any other medium. Insuring, as this plan does, all the operatives of a manufacturing establishment, it gives opportunity for study of actual mortality from the standpoint of the inherent dangers in the occupation both as a direct and as a contributing cause. The experience furnished through this medium will be

subject to the inaccuracies caused by the shifting and instability of labor but it will be possible to overcome this in large measure. For instance, a separate class can be made for purposes of statistical analysis of operatives who have been for a number of years in the same plant, and thus the factor of inaccuracy caused by floating labor can be largely done away with.

Insurance companies have, on the whole, showed the greatest tendency to get together and to compare results of any of the great forces working on the problem of hazard to life. From the standpoint of the life insurance companies there have been two notable examples of this. The specialized mortality investigation of 1903, conducted by the Actuarial Society of the United States, was participated in by thirty-four life insurance companies, each furnishing its quota of exposures. It is of no particular value as applied to unhealthy trades, principally because few of the companies involved had any such exposures to contribute. The only occupations considered, which might be said to involve an industrial disease or which were inherently unhealthy, were steel grinding, glass working, pottery making, house painting and printing, and in most instances the classes were so small that the information could not be depended upon. Other classes of occupations in which the hazard was accidental were taken up and valuable results obtained. In 1913 the Medico-Actuarial Mortality Investigation was completed. This study was conducted by the Actuarial Society and the Association of Life Insurance Medical Directors of the United States; was participated in by forty-three first-class companies, and provided an immense amount of vital data. From the industrial disease standpoint the information obtained, while somewhat more comprehensive than provided by the specialized investigation of 1903, was, in general, subject to the same criticisms as applied there. The classes were generally small and liable to fluctuation. The industries investigated which have a bearing on our problem were: journeymen baking, glass industry, hatmaking, metal grinding and polishing, blast furnaces and rolling mills, coal and metalliferous mining, potteries, house painting, plumbing and steam fitting, printing, stone cutting and tanning. This list does not, of course, even scratch the surface of the industrial disease problem. More work of this character, however, may be expected in the future, and with the spread of life insurance through all industries should have far reaching results.

Although not strictly to the point, some mention should be made of the educational effect of insurance selection on industry. This has been demonstrated in the realm of safety through the liability companies and their various rating boards, which fix a rate according to actual safety conditions in individual plants. Work of this character must also be done in relation to disease and poison hazards by the companies who do group insurance. But with the extension of insurance on the individual to industries whose operatives have, in large measure, been deprived of its benefits, will come an education of the individual worker which should have a far-reaching effect. There may be said to be roughly two methods of selecting life insurance risks from the standpoint of occupation; the individual method, and the class method. By the class method, the particular plant in which the individual works is not taken cognizance of at all and the job is rated the same everywhere. By the individual method, an effort is made to distinguish between plants having up-to-date conditions and those in which conditions are old-fashioned and bad. This is sometimes done by dividing the risks into two or three classes and charging a different rate for each. If Jones who works in Factory A is charged more for his protection than Smith who works in Factory B, both being at the same job, the education of Jones as to the necessity of good working conditions is at once accomplished. As yet this particular method of selection is in its infancy and life insurance companies make very few industrial investigations; but with the development of the new lines of insurance, and with the incorporation into the policies of the special benefit clauses, and with the general interest manifested in the study, a great deal of intensive work along the lines of industrial health is inevitable in the future.

Practically all the great manufacturing companies are to-day keeping health records of their employees and, in many

instances, these records are the most valuable data obtainable on the subject of industrial diseases. Of late years, the tendency to keep these records in a thoroughly systematic way has been growing with leaps and bounds. The laws compelling the reporting of industrial diseases already in force, and the proposed laws forcing the payment of compensation for industrial disease, will make the practice of keeping accurate industrial disease records universal. The growing interest in the problem of scientific employment management will assist in bringing about this result. One of the principal features of a proper employment department is its record system. Here a rigid tab is kept on the health of the plant. This is probably primarily to check up on causes of absenteeism but, if the records are properly made and kept, will aid greatly in showing the prevalence of industrial disease. It has often been noted in plants that do not maintain separate employment departments, that men will stay out for a day or so for illnesses which are undoubtedly due to industrial causes, such as incipient lead poisoning, and yet never report to the plant medical department. Such cases would instantly be detected by the employment department and the operative forced to report to the plant physician.

The federal government during the war gave the campaign for scientific employment management its hearty support. The great army departments, ordnance, quartermaster, aircraft, etc., maintained industrial service sections which preached the doctrine unremittingly. Skilled men were sent out who aided in the formation of properly organized employment departments in plants working on government contracts, and in the great government-owned plants. Courses in employment management were instituted in the universities, and men and women trained to organize and take charge of such departments. Such organizations in plants, working in close co-operation with the medical departments and the safety departments, will undoubtedly produce a mass of data of incalculable value.

With all the agencies which have been briefly mentioned above at work gathering data, it might be supposed that the problem approached a final solution. As a matter of fact, we might approach a very good estimation if all information now being gathered from all sources could be collected and thoroughly digested. There are at the present time a number of federal bureaus and departments which are doing much valuable work along this line. Of course we are all familiar with the work of the Bureau of Labor Statistics of the Department of Labor, and it is safe to say that this bureau has done more in collecting information from differing sources than any other agency. During the war other services were organized which should be perpetuated. It is undoubtedly true that the federal government can do more toward a unity of effort in the solution of any given industrial problem than any other agency, and unity of effort is what is needed more than anything else in the solution of this one.

The Working Conditions Service of the Department of Labor stands in an excellent position to unite all interests concerned with any problem of industrial welfare. It maintains separate departments dealing with industrial hygiene, safety and employment management; it stands in a position to make inspections and to give expert advice and counsel on industrial problems at the call of any organization; it works in close relation to the United States Public Health Service, and the personnel for its inspection and industrial hygiene work is furnished by that department.

The Working Conditions Service is also in constant co-operation with the great government departments which have to do with the industrial problem, such as the Bureau of Mines, the Bureau of Standards, Army and Navy Ordnance, Quartermasters, as well as the Departments of Labor of the individual states. The Bureau of the Census has contributed much valuable information to the subject. The mortality statistics of 1909 were particularly valuable from the standpoint of accident and gave some information from the industrial disease standpoint which will be further mentioned later on.

No better illustration can be given of the difficulties attending even a rough estimate of the rise in mortality caused by an unhealthy trade than a brief outline of some of the data on lead poisoning. This is selected because it is the most prevalent

of all the industrial diseases, many times the number of deaths from lead poisoning being reported than from all other occupational poisonings combined.

The mortality statistics of the Bureau of the Census give the number of deaths annually from lead poisoning. These figures include only about two-thirds of the United States, the rest being outside the registration area.

The table for the years 1910-1916 follows:—

Year	Percentage Registration Area Population to Total United States Population	Number of Deaths
1916	70.2	190
1915	67.1	155
1914	66.8	149
1913	65.1	162
1912	63.2	148
1911	63.1	145
1910	58.3	136
		1085

This table shows that 1085 deaths during the seven years of 1910 to 1916, inclusive, were due to chronic lead poisoning.

In Great Britain during the five years of 1910 to 1914, inclusive, there were reported 2,741 cases of lead poisoning with 174 deaths from this disease or a percentage of fatal cases of 6.3. (The British law provides for the reporting by physicians of cases of lead poisoning in certain lead industries. Painting, which accounts for a great many cases of plumbism, is not covered by the law.) Assuming that the same proportion of fatal to total number of cases holds for the United States, we might roughly estimate that the 804 deaths in the United States registration area during the five years, 1912-1916, inclusive, represent between 12,000 and 13,000 cases of plumbism (exact figure calculated is 12,762).

Localized studies of lead poisoning have been made from time to time. Dr. Alice Hamilton in Kober and Hanson, *Diseases of Occupation and Vocational Hygiene*, gives the following figures:

The Commission on Occupational Diseases in Illinois discovered 578 cases that occurred during the three years, 1908-1910. These were scattered through seventy occupations. Pratt, in a study for the New York State Factory Investigating Committee, discovered 376 cases of lead poisoning which had occurred in New York City during 1909, 1910 and 1911. An investigation into the white and red lead industries in 1911 showed 388 cases of lead poisoning during sixteen months in factories with a pay roll of 1600; sixteen of these cases were fatal; eighty-nine out of 120 of these cases became ill after less than one year's work. Considering the time of this investigation to be practically a year and a half the exposures would be 2,400. This would give a death rate of 6.7 per 1,000 exposed, from chronic lead poisoning alone, taking no cognizance of cases in which it was a contributing cause. This would represent a death rate approximately seven times as great as that from tuberculosis in a healthy locality.

Dr. Hamilton in the same article states the result of further investigations as follows:

Art and Utility Ware: 13 cases among 85 dippers in one year. Among dippers' helpers there were 13 cases among 180 men and boys, and 29 cases among 150 women.

White Ware: 796 employed; 60 cases in two years.

Art and Utility Ware: 304 employed; 63 cases in two years.

Tiles: 304 males employed; 48 cases in one year: 243 females employed; 28 cases in one year.

Bath Tubs: 1012 men employed; 217 cases in 1911.

Lead Smelting and Refining: 1667 cases in one year in nineteen plants employing 7400 men.

Storage Battery Manufacture: 164 cases in 1913 in five plants employing 915 men.

The deaths are not given but we may compare the frequency rate with Dr. Hamilton's figures on the white and red lead industries.

Industry	Frequency Rate per 1000 Exposed
White and Red Lead	243
Art and Utility Ware (Dippers)	153
White Ware	38
Art and Utility Ware	105
Tiles (Males)	158
Bath Tubs	214
Smelting and Refining	225
Storage Batteries	168

The highest rates are where one would expect to find them, with the exception that one would naturally imagine a somewhat higher rate in storage battery plants. In all probability the death rate would follow in the same proportion.

The Occupational Disease Act of Illinois provides for a monthly physical examination of the employees in certain specified industries considered hazardous. For the year ending June 30, 1917, 150 firms were reported on. The kind of plants and the number of cases of lead poisoning discovered in each are given in the following table:

Industry	No. of Firms	No. of Cases
Painting	53	22
Smelting and refining	35	104
Paint manufacturing	26	5
Storage battery manufacturing	10	3
Manufacturing of tinware	6	10
White lead	4	2
Electrotyping	4	3
Manufacturing car seals and bearings	4	..
Telephones and switchboards	4	..
Wallpaper and oilcloth	3	..
Enameling	1	..
Total	150	149

The average number of men examined for the year in these 150 plants was 6,497. The 149 cases of lead poisoning found give a rate of 2.3 per 100 employed for the year.

In New York state, there were reported for the six years ending August 31, 1917, 617 cases, ninety-one of these resulting in death.

More than half of these 318 cases with fifty-nine deaths occurred among the painting trades. The other 299 cases and thirty-two deaths occurred in over thirty-three occupations and industries. The following table gives the more important industries and occupations from the point of view of the number of cases reported therein:

Industry	No. of Cases	No. of Deaths
Painting	318	59
House, structural and ship painting	246	48
Automobile, carriage and wagon painting	45	7
11 other industries	27	4
Storage battery manufacturing	122	3
White lead, paint and colors	55	4
Printing	16	4
Smelting or casting lead, solder, etc.	15	3

All the investigations into lead poisoning reveal the fact that painting is responsible for a large proportion of the cases discovered. The New York state figures just mentioned show that 318 cases out of 617 were among painters. Of the 578 cases investigated by the Illinois Commission, 157 were painters. Forty-two painters were found among 109 cases investigated by Pratt in New York City. Andrews quotes a hospital record in which twenty-eight out of fifty-nine cases were painters and of his sixty fatal cases, forty were painters. In 1913 Hayhurst examined 100 able-bodied painters and found fifty-nine with evidence of plumbism. The figures for Great Britain confirm the American data in regard to lead poisoning among painters.

The law requiring the reporting of occupational diseases in certain specified industries does not apply to house painters. Nevertheless, physicians frequently report cases of lead poisoning among house painters. One thousand, nine hundred and seventy-three cases among house painters and plumbers were reported in the ten years, 1900-1909, inclusive, and 232 cases in 1910, 197 of which were in painters and thirty-five in plumbers. In the ten years, 1900-1909, there were 387 deaths from plumbism among painters and plumbers. Assuming that the proportion of deaths to the number of cases is the same among house painters as among other lead workers, Dr. Legge estimates that over 9,000 (exact figure 9,516) cases occurred among the former during the years, 1900-1909, inclusive. While in the thirteen year period, 1900-1912, inclusive, only 8,523 cases occurred among the reportable occupations, during the fourteen year period, 1900-1913, inclusive, there were reported 421 deaths among those occupations coming under the jurisdiction of the law and in the same period there were reported 427 deaths among house painters and 129 among house plumbers due to lead poisoning. On the basis of the British census returns and the estimated number of cases of plumbism among painters, Legge estimates an attack rate among these workers of 4.8 per 1,000 per annum.

The actual data contained here when viewed as the basis of a mortality rate is exceedingly limited. It shows the undoubtedly high rate of prevalence of the disease. Owing to the very great number

of those engaged in the painting trades, the general figures are confusing. Dr. Legge's estimated attack rate seems remarkably low but probably a large majority of cases are never reported.

The life insurance figures are of very little value. The Medico-Actuarial Investigation showed a mortality of 111 per cent. of the expected for journeymen house painters, and in the printing trades 102 per cent. of the expected for journeymen compositors, and 117 per cent. of the expected for journeymen pressmen. The Metropolitan Life Insurance Co., Industrial Department, Mortality Experience, 1911-1913, inclusive, (occupied white males, ages fifteen and over) shows eighty-one deaths due to chronic lead poisoning, fifty-four of which were among painters.

Practically all of the data, except the United States census figures, are at least eight years old and it must be admitted that industry has made great strides in the last ten years. The United States census figures show a constant increase in the number of reported deaths. This is undoubtedly due to the extension of the registration area, more correct reporting of the cause of death, and increase in the number of operatives engaged. The only year in which we can get a comparison of the deaths as reported by the Bureau of the Census and the approximate number of operatives exposed to the poison is 1910. During that year there were 136 deaths from chronic lead poisoning. The census report for 1909 gives an industrial classification, and we can approximate roughly the number of operatives engaged in trades which expose them to the disease. This is estimated at about 1,000,000. A further approximate separation into the group which is actually exposed to the poison would give about 450,000. Of these, 337,000 were painters and must be treated separately.

To eliminate painters, we must ascertain as nearly as possible their proportion of the deaths. From all the figures quoted above and also from the census returns of previous years, this can be estimated at about 50 per cent. This leaves us, then, 113,000 operatives and sixty-eight deaths. In our group of operatives we still have 47,000 representatives of the printing trades, a group which can be removed as its mortality is small from this cause. A study of the proportion of deaths to be ascribed to the printing trades, shows five to be a probably correct allowance for the year. We have therefore remaining 66,000 operatives and sixty-three deaths, or less than 1 per 1,000 exposed. The actual death rate, even by this calculation, is too small as the figures on the deaths merely represent the area of registration, while those on the trades are taken from the whole country. All the great industrial centres, however, are within the area of registration except the state of Illinois, and in that state the principal industrial centres are within the area.

This study is, of course, filled with inaccuracies and open to every kind of criticism. It bears, however, a certain similarity to Sir Thomas Oliver's figures on mortality among pottery workers exposed to the poison. Eleven years' experience, 1899-1909, inclusive, as published in this report, showed a death rate of about 1 per 1000 exposed.

Taking painters and varnishers separately, the rate sinks to infinitesimal dimensions, even allowing for an enormous dilution. A report published in August, 1918, by Dr. Louis R. Harris, director, Bureau of Preventable Diseases, New York City Department of Health, gives a study of 402 painters. A rate of prevalence of active or latent plumbism of 40 per cent. is reported among this group. Compared with Dr. Legge's estimated attack rate of 4.8 per 1000 these figures are remarkable.

From all the data available we may make the following deductions:

1. The actual mortality from chronic lead poisoning is small, even making all possible allowance for dilution of exposures by lives not dangerously in contact with the poison.

2. We must look to lead poisoning as a contributing cause in ascertaining the actual increase in death rate from this cause, as the rate of prevalence is undoubtedly high.

3. In certain groups such as the white lead workers, as found by Dr. Hamilton, mortality may run high from the direct cause.

4. Painting is not so dangerous a trade as working directly in lead compounds, as occurs in white lead works, glaze rooms in

potteries, smelting and refining works and storage battery plants. The immense preponderance of cases reported in the painting trade is due to the large number of workmen in this trade and to the fact it is a skilled, well-paid trade at which the workers remain, thus eliminating the factor of instability. On the other hand, it is easier to remove or mitigate the dangers in a lead plant than it is in the painting trade, because in the latter instance the amount of exposure is wholly dependent on the discretion of the individual worker.

Miners' consumption ranks on a par with lead poisoning as the most dangerous industrial disease. The information bearing upon it is not so confused and liable to error as the data upon lead poisoning, for the reason that it is a better paid occupation in the main than are most of the lead trades, and because the miner remains rather constantly at his trade. A small amount of fairly direct information is therefore obtainable. As has been pointed out many times, and especially shown by Lanza in his Joplin report, this disease is principally dangerous when the mining is done in silicious ores. In this, as in lead poisoning, the actual rise in mortality must be looked for from other immediate causes than miners' consumption, principally from tuberculosis and pneumonia.

The insurance figures can be considered first. The Medico-Actuarial Investigation found a mortality of 226 per cent. of the expected, according to the mortality table, among miners, other than coal miners. This means, of course, that practically the entire class was composed of metalliferous miners. The class was large, showing 48,400 exposures. The accident mortality was seven times the standard; pneumonia, three times the standard; and tuberculosis, four times the standard. The class included all metalliferous miners and therefore there was some dilution in so far as the effects of silicosis were concerned.

Studies of individual life insurance companies have shown mortalities of over 300 per cent. of the expected and with the death rate from pneumonia and tuberculosis running seven to ten times the average. Mortalities in individual years have been known to go as high as 450 per cent. of the expected.

A study of the health records of a big mining center, where the operations were all in silicious rock, for four years, 1906, 1907, 1908, 1909, showed the following deaths from tuberculosis and miners' consumption among miners: 1906, 59; 1907, 65; 1908, 76; 1909, 82.

In 1911 the figures were checked for six months of that year and found to be running in about the same proportion.

The number of miners in the district was about 11,700 (estimated). The death rate per 1000 from this cause for the four years was therefore:

1906	5.04
1907	5.55
1908	6.49
1909	7.01

Dr. Frederick L. Hoffman in his study of mortality from respiratory diseases in dusty trades, published by the Bureau of Labor Statistics in 1918, gives a study of the mortality among copper miners of Montana for the years 1907-1914, inclusive. The figures given there are prepared with the co-operation of the local Board of Health of the city of Butte. Estimating the number of miners in the Butte district at about 15,000, Dr. Hoffman's eight-year study would return a death rate from respiratory diseases of about 9 per 1000 miners.

In 1904, Dr. J. S. Haldane, F.R.S., made an investigation into miners' phthisis among the metalliferous miners of Cornwall. Dr. Haldane found the death rate from lung disease for three years, 1900-1902, inclusive, as follows:

Age	20–25	25–35	35–45	45–55	55–65
Death Rate	2.7	17.3	33.2	32.2	42.6

These figures run higher than the average of definite American figures, but they are probably more free from dilutions.

Dr. A. J. Lanza of the United States Public Health Service, in his report made January, 1917, estimates a rate of prevalence of 30 per cent. for the disease in the Joplin district.

We can estimate a death rate of from 7 to 10 per 1000 employed, from lung disease induced by the employment, in districts where hard rock mining is carried on. Add to this an accident mortality of about 4 per 1000 engaged, and you have a death rate from hazards wholly incident to the occupation of from 11 to 14 deaths per

1000 employed. These figures are minimum. It is probable the rate runs higher. The average death rate by the American Experience Table of Mortality runs from about 8 per 1000 at age twenty-six, to about 10 at age forty-one. From this the rise in mortality from this particular trade can be readily seen. A very small part of the available material is given here, since the subject has been so thoroughly covered by Dr. Hoffman and Dr. Lanza in their recent papers already referred to.

Of course, the death rates suffer a tremendous diminution when modern methods of ventilation and sanitation are introduced; when the use of water-fed drills supplants the dry, and when modern change houses are built. The very much more definite information as to the actual hazard in mining as compared to the lead trades has been largely accomplished through the close co-operation of a number of the forces working on the problem. It is to be hoped that in the future the principle of co-operation of all interested forces, which was so much emphasized in Washington during the war, will be applied, for the common benefit of all, to the problem of lessening the morbidity and mortality of industrial workers.

THE PATHOLOGICAL AND CLINICAL MANIFESTATIONS FOLLOWING THE INHALATION OF DUST*

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"Failure to inquire respecting the occupation, and a want of knowledge of its working condition and customary ailments, places the doctor at a disadvantage toward his patient, and deprives the latter of the full benefit of treatment."—*Arlidge.*

INTRODUCTION

PNEUMOCONIOSIS occupies a curious position. The term is a familiar one and the nature of the disorder is described in all text-books dealing with diseases of the lungs. In spite of this, however, the term is rarely used by the clinician and only a little less frequently by the pathologist. Furthermore, one may search statistics dealing with morbidity and mortality rates and never find any mention of the condition. In those instances in which the deposition of dust in the respiratory system has actually given rise to pathological changes, the illness or the cause of death is ascribed to secondary changes, such as chronic bronchitis, asthma or tuberculosis. The etiological factor is thus concealed by the use of other terms. The reason that mild grades of pneumoconiosis are rarely mentioned as such is to be ascribed to the fact that a slight degree of pneumoconiosis is present in the lungs of all city dwellers who have reached adult life, the condition under such circumstances being regarded almost as normal. Furthermore, in the presence of the mild type or in the very early stages of the serious and progressive type there are no symptoms, nor is there anything abnormal to be found on physical examination. In the advanced stage the condition is usually referred to as fibrosis or cirrhosis of the lung. Now that the X-rays have come into general use, we have a ready method of determining the condition almost from its incipiency. In the future, pneumoconiosis, with or without fibrosis, will probably be used more generally as a distinctive term. An additional reason for bringing it more prominently to notice is the ever-increasing interest in diseases associated with certain occupations. There can be no doubt but that the whole subject of dust in its relation to disease is in need of more intensive study than has been given to it in the past. Collis (1) has stated that until recent years the tendency has been to consider, as a broad truth, that dust inhalation predisposes to diseases of the chest, of which pulmonary tuberculosis is the chief. "Closer investigation," he

*Received for publication April 26, 1919.

continues, "is proving that the subject is more complicated; that respiratory diseases caused by dust vary with the nature of the dust inhaled; that although a special type of pulmonary tuberculosis occurs upon the inhalation of certain dusts, it is not associated with the inhalation of all kinds of dust; and that other forms of chest disease, which, though not equally fatal, seriously impair the respiratory organs, are set up by inhaling special forms of dust."

Moreover, as has been pointed out by Pometta (2), it is to be borne in mind that there is scarcely any sickness from industrial causes that cannot develop, with about the same group of symptoms, from some entirely different cause.

The first systematic treatise on the effects of the inhalation of dust was contributed by Ramazzini (3) in 1700. From that time down to the latter part of the nineteenth century, the literature contains numerous references to the subject. Some call attention to the association of certain occupations with respiratory disorders, others have to do with the pigmented areas in the lungs and their possible association with dust. In 1838 Stratton (4) suggested the name anthracosis to designate the changes produced by the inhalation of coal dust. Since that time a number of terms have been used to indicate the particular variety of dust, namely, silicosis, siderosis (iron dust), etc. In addition a variety of popular names have been applied to the dust diseases, the terms used indicating the occupation, as for example, miners' asthma, black-spit, grinders' rot, potters' rot, potters' asthma. The preferable term is that suggested by Zenker—*pneumoconiosis* (πνεύμων, lung; κονις, dust).

Among the earliest observations may be mentioned those by Wafler (1729) and Le Blanc (1775) on the great mortality among the makers of French millstones; and Johnstone (5) on the mortality among the needle pointers of Worcestershire. Thomas Beddoes (6) quotes Linnæus to the effect that cutters of grinding stones all die phthisical before the thirtieth year. Beddoes also noted the frequency of this affection among stone cutters, needle grinders, brass workers and miners, but states that others, as japanners, millers, etc., are not subject to this disease. Beddoes was far ahead of his time in expressing the belief that the high death rate from phthisis among textile workers, tailors, glovers, etc., was to be attributed to confinement and inactivity rather than to dust.

In 1813, Pearson (7) first suggested that dust particles gained access to the lung tissue, and he attributed the dark pigmentation often met with in the lung to the actual deposit in it of particles of carbon or soot derived from the air. Similar observations by Christison (8) and Graham (9) confirmed this view. Virchow, however, denied that the pigmentation was due to carbon and attributed it to organic pigment from the blood. Among those who furnished the proof that the dust actually was deposited in the lung may be mentioned Traube (10), who discovered in the lung of a charcoal burner the angular particles of charcoal dust; Zenker (11), who showed by chemical tests the presence of iron dust and Knauff (12), who demonstrated by inhalation experiments particles of soot in the lungs of dogs. Finally in 1866 Virchow (13), himself, acknowledged that the proof was indubitable.

One of the most interesting and unique studies of occupations is that by Henry Mayhew entitled *London Labor and the London Poor; a Cyclopædia of the Conditions and Earnings of Those That Will Work, Those That Cannot Work, and Those That Will Not Work.* This book, published in 1851, is the first of which I have any knowledge to portray the life of the worker from every standpoint, namely, the character of his work, his hygienic habits, his amusements and his environment—both at work and at home.

Until the discovery of the tubercle bacillus by Koch, in 1882, no distinction was possible between extensive non-tuberculous and tuberculous fibrosis. The term fibroid phthisis was therefore very loosely used and, even at the present time, many cases of non-tuberculous fibrosis are classed as instances of tuberculosis. Tatham (14), in commenting on this aspect of the question, states that "it is certain that much of the so-called potters' phthisis ought properly to be termed cirrhosis of the lung. Deaths from this affection should never be included under the head of phthisis, which term is now restricted by universal

consent to the tubercular malady of that name."

Among others who have written largely on the subject may be mentioned Thackrah (1832), G. Calvert Holland (1843), Arlidge and Oliver in England; Hirt (1871) in Germany; and Hoffman and Kober in this country.

ETIOLOGY

As the result of the rise and expansion of a wide variety of manufacturing interests during the nineteenth century, the attention of sanatarians became more and more directed toward the harmful effects of industrial processes. And of the various harmful agencies none has received more attention than dust. In England, because of the number and variety of her manufacturing interests, the subject has received a great deal of attention. In this country but little thought has been bestowed on the subject until within the past decade. But if we have been backward in realizing the importance of the subject, there is no doubt that the period of apathy has passed. As Hoffman recently remarked, "no nation within recent years has equaled us, either in the quantity or the quality of the work done along the lines of industrial medicine." Much as is known regarding the effects of dust, there is still much to learn and, more important still, there is much to unlearn. At the present time there are too many statements made for which there is no adequate proof. In not a few instances statements regarding the harmfulness of certain dusts have become traditional and continue to be accepted by each succeeding writer on the subject. Many such statements are subject to revision. At the present time far too much of our knowledge regarding the evil effects of dust is based on mortality statistics. While we have no desire to discredit or belittle data of this sort, it is undoubtedly true that mortality returns give a very false idea in many instances, particularly in reference to the organic dusts. As a result of studies of this kind many industries have gained an evil reputation because of a high death rate from tuberculosis among the employees. It has been assumed by most writers that the dust is unquestionably the exciting cause of the tuberculosis. In nearly every instance this assumption is not susceptible of proof. I shall go into the subject of tuberculosis and industrial dusts more in detail in another place.

In studying the effects of dust on the health of the workers, the following factors must be kept in mind: (1) The type of dust; (2) The trade process followed by the worker; (3) The sanitary history of the worker, both personal and in the home.

Type of Dust

That dust is of very varied constitution is well known. First, it may be divided into two main parts, namely—inorganic and organic. The former may be farther subdivided into metallic and non-metallic, while the latter is distinguishable into animal and vegetable. To these two main divisions Hirt (15) added a third, namely —mixed dust.

Inorganic Dust

The inorganic dusts vary greatly in their physical characters; thus the particles may be acicular, or jagged in outline, or oval, or circular. The more jagged and angular the particles, the greater is their power of mischief. Another factor to be considered is the chemical composition of dust. Arlidge (16) has emphasized the importance of density and chemical composition. For instance, density is of great importance; and so is the degree of their divergence as regards organization and chemical constitution. Collis (1) has expressed the opinion that in general dusts appear to be more injurious as their chemical composition differs from that of the human body, or from the elements of which the body is normally composed. Thus it is seen in the case of lime dust (a normal constituent of the body) that notwithstanding its angular form, it is but a feeble irritant to lung tissue. Another example of comparative innocuousness of mineral dust is found in plaster of Paris in its native state. Although its particles have a square or rhomboidal form, with more or less acute angles, they have not the gritty hardness of silicious dust and diffuse readily through water.

In our own studies, cement dust, in spite of the enormous quantities inhaled, seems to be relatively harmless. This is probably due to the fact that it contains little or no silica.

Most of the recent studies on inorganic dust make it clear that metallic and siliceous dusts are the most potent causes of pulmonary fibrosis. The latter is by far the most important because more frequently encountered. The second report of the British Royal Commission on Metalliferous Mines and Quarries (1914) quotes experiments of Beattie as follows:

> "Certain mineral dusts, such as coal, clay, cement, were not shown by experiment to be injurious. Other dusts, e.g., silica, quartz, flint, sandstone, are injurious, as are also carborundum and emery. After careful consideration, therefore, we feel justified in concluding that, even although further investigation should disclose other dusts as injurious, the dust of fine crystalline silica is especially injurious and is the more potent cause of fibrosis."

The commission was further of the opinion that inorganic dusts may be grouped in two classes:

> (1) "Dusts, the inhalation of which has not so far been shown to be associated with any marked increased mortality from respiratory diseases—to this class belong coal, shale, slate, iron ore, clay, limestone, plaster of Paris, and cement." (2) "Dusts, the inhalation of which is associated with excessive mortality from respiratory diseases, and especially from phthisis—to this class belong quartz, quartzite (i.e., ganister and buhr stone), flint and sandstone."

It seems reasonably certain, from the evidence at hand, that hard, gritty dusts composed of sharp and jagged particles are the ones to be especially feared. The least innocuous of the inorganic dusts is soot. In most large cities and especially those containing large manufacturing plants, the air is filled with soot. Klotz (17) in a study of the lungs of individuals long resident in Pittsburgh, Pa., has shown that a mild grade of pneumoconiosis and fibrosis arises from this source.

The evil effects of metallic dust, usually in the form of fine particles of steel or iron, are frequently referred to and are cited by most writers as the most dangerous of all. It is noteworthy, however, that fine iron particles have rarely, if ever, been demonstrated in the substance of the lung. The cases of siderosis reported by Zenker (18) and Merkel (19) were caused by the red oxide of iron used by gold beaters and looking-glass polishers. The first case observed by Zenker was a girl who had inhaled a thick dust of iron for ten or twelve hours a day. Her work was to color blotting paper with a powder of red oxide of iron. Up to 1912, twenty-one cases of pneumoconiosis of this type had been reported (Dieulafoy).

Greenhow (20) in the examination of a grinder's lung, could find no excess of iron, either chemically or by the use of a magnet. In common with a number of other observers he was of the opinion that the pulmonary lesions were to be attributed to an excess of silica from the grindstone. Soper (21) in a chemical analysis of the dust from the New York subway showed that it contained 61.30 per cent. of iron, nearly all of which was in the metallic state; there was also 15.58 per cent. of silica. He points out, however, that it must not be supposed that the aggregate weight of metal which these figures indicate was capable of being mixed with the air which is breathed. Many of these iron particles were visible to the naked eye and too heavy and too large to be carried far or be sustained in the atmosphere, even by the strongest draughts of air.

The *size of the individual particles* plays an important part. The finer the dust the more dangerous, and there is every reason to believe that only the very finest particles obtain a permanent footing in the pulmonary tissue. Collis (1) lays special stress on the fineness of the dust, as in his opinion, it is only the very minute particles which gain access to the lungs. He states that all materials similar in origin and in chemical composition do not give rise to dust of similar fineness, and in this element of difference may lie the explanation of the fact that two otherwise similar dusts may produce dissimilar symptoms.

Several excellent studies have been made on the size of the silica particles found in the lungs. Watkins-Pitchford (22) states that the majority of visible siliceous particles (as detected by means of polarized light), in the sections of silicotic lung vary from 2 μ to 12 μ in maximum diameter. Larger particles seen by him in his investigation were found, not in the lung tissue, but in the cavities of bronchioles or alveoli. John McCrae (23) studied six lungs of miners who had suffered from silicosis. The ash from each lung was carefully dried at a low temperature so that no splintering of the silica particles should take place by excessive heating. A thin emulsion was then made from the powder and examined with the microscope. The great majority of the particles

were found to be of indefinite shape and to have a diameter less than 1 μ (that is, a diameter less than one-eighth of that of a red blood corpuscle) ; the amount of material with a diameter less than 1 μ was very approximately 70 per cent. of the whole. Of the remaining 30 per cent. the longest diameter varied between 1 μ and 8.5 μ. Only a negligibly small number of particles was seen whose longest diameter exceeded 8.5 μ, and the very longest observed was 10.5 μ.

Watkins-Pitchford (24) found that the prevailing outline of the particles (as shown by polarized light) was narrow and elongated with one or both extremities pointed; many showed a slight curve in their long diameter and some were elliptical; the commonest form was that of a very slender prism, having its ends cut off at a right angle or obliquely.

Concentration of the dust is also an important factor. If the worker is exposed to dust in a closely confined space the effects are more severe and pathological changes are more quickly produced than if the exposure occurs in a well-ventilated room or in the open. The very severe effects produced in the gold miners of South Africa and the zinc miners in southwestern Missouri are largely due to the fact that the rock dust accumulates in underground passages and chambers. In the Rand, holes are bored into the rock, which, if not moistened, emit a constant stream of fine dust which thoroughly impregnates the atmosphere; and, after blasting, sufficient time is not allowed to permit of the dust settling. The finest dust is produced by blasting; over 99.5 per cent. of the particles are below 12 μ in diameter and the average diameter is under 2.5 μ. This dust is produced in enormous quantities, although only over a short space of time. The dust contents of the air after blasting may amount to 500 mg. per cubic meter and contain in that volume nearly 86,000,000,000 particles of a diameter of 12 μ and downwards (25).

The length of exposure is also an important factor. It is rare, for instance, to find the appearance of definite early changes after exposure to siliceous dust with an exposure less than four or five years (Watt and Steuart). In the case of dusts less injurious, even early pulmonary changes may not be detected under ten or even fifteen years of exposure.

Briefly summarized, inorganic dusts, no matter what their source, produce definite changes in the lungs. They differ in the severity of the lesions produced in accordance with hardness, sharpness and chemical composition. At one end of the scale are the siliceous dusts, capable, under favorable conditions, of producing serious pulmonary damage in a comparatively brief period of time. At the other end are those which produce slight changes and then only after years of exposure. Among the latter may be mentioned plaster of Paris, lime, bituminous coal and soft carbonaceous material in the form of soot.

Organic Dust

Whether organic dust is of itself injurious is debatable. Certainly up to the present the evidence in favor of its causing chronic pathological changes in any way comparable to those produced by inorganic dust is not forthcoming.

The inorganic dusts are in part animal and in part vegetable. It is conceivable that a few of them, such as the dust arising from very hard wood, such as ebony or rose wood, may cause damage. And the same may be said of certain of the animal dusts such as ivory, bone, tortoise-shell, and horn. But even these stand very low in the list of injurious dusts and it is questionable whether the dust involved by working these substances is sufficiently fine to cause trouble.

Of the other organic dusts, some vegetable, others animal, which are encountered in the textile manufactures, there is no proof that they cause changes in the lungs. Among these dusts may be mentioned cotton, silk, wool, flax, hemp, hair, tobacco, etc. We have already seen that even the inorganic dusts vary tremendously in their power for mischief and that their injurious properties are largely dependent on hardness and on the angularity of the individual particles. If these properties are lacking the resultant injury to the respiratory tract is slight or even negligible, as in the case of soot or cement dust. Such being the case it is difficult to see how dust which is utterly devoid of these properties can of itself cause injury. As we shall show later, manufacturing processes associated with organic dust have in many instances been given a bad

name because of the high incidence of tuberculosis among the employees. It has been assumed that the dust was responsible. This I disagree with and my reasons for this will be given in another place.

Oliver (26) is of the opinion that dust composed of cotton fibres is *per se* innocuous, and if the moisture necessary in the weaving of cotton could be dispensed with the trade would be harmless. On the other hand Collis (1) states that cotton "strippers" who are exposed to much cotton fibre dust often develop, after some years of exposure, a condition in which the worker experiences asthmatic symptoms and sense of tightness across the chest, but that phthisis is not found to be especially prevalent among those affected. Oliver also is the authority for the statement that he had never seen any ill effects associated with the manufacture of cigars or cigarettes, at least in so far as dust is concerned. The experience of Dr. Miller and myself is in agreement with this.

In this study Drs. Miller and Smith have shown that the dust obtained from places associated with the organic type was never purely organic but a mixed form containing greater or smaller quantities of inorganic material. And even where the inorganic is present in noticeable quantities it is not sufficient to produce changes in the lungs which differ in any particular from those observed in the urban dweller. For instance, the investigation of a carpet manufactory revealed an appalling state of dustiness, and yet in spite of exposure for many years the roentgenologic pictures of the lungs showed changes which differ in no respect from those encountered in individuals who have lived all their lives in a large city.

This is in striking contrast to the changes observed among those exposed to inorganic dust. In these individuals the changes are definite and they show a steady development; so much so that, given the type of dust and the length of exposure, one can hazard a shrewd guess as to pulmonary changes as revealed by the roentgenograph.

If organic dusts are incapable of producing chronic inflammatory changes in the lungs, can they be wholly disregarded? In the light of our present knowledge this would be unwise. We need more and better studies in regard to this type of dust with reference especially to the part it plays as a carrier of bacteria. In order to do this intensive studies are needed. All contributing factors must be eliminated, and this is especially true in regard to tuberculosis. Faulty hygienic conditions in the shop; unsanitary living conditions; malnutrition and insufficient wages must be eliminated as factors.

Recent studies on the etiology of asthma point very clearly to the fact that the disease is, in many instances, apparently an evidence of protein hypersensitiveness. In many instances the evidence seems clear. Among the better known examples are instances in which an asthmatic attack is brought about by exposure to the emanations of animals or the inhalation of dust containing pollen. The condition is analogous to that produced in animals by the injection of a foreign protein and is characterized by spasm and constriction of the finer bronchioles and, to a lesser extent, by swelling of the mucous membrane of the bronchioles.

In several occupations associated with organic dust, acute respiratory symptoms often arise in susceptible individuals which are probably the result of protein poisoning. Oliver (27) states that in the manufacture of shoddy the grinding of the rags by machinery results in the formation of considerable quantities of dust, the inhalation of which is very trying to new hands but which has little or no immediate effects on the older work people. Those persons who are new to the trade develop what is known as "shoddy fever," the symptoms of which are a rise in temperature, severe headache, signs of bronchial catarrh, and running at the nose. The work people shiver as if they were going to have a severe fever and they complain of muscular pains. The condition is sometimes referred to as "mill fever" and has been noted as occurring in cotton operatives and those who work in flock, jute and flax. A similar condition is observed in grain threshers. Workers who are especially susceptible will, in the beginning of the threshing season, develop a headache and great irritation of the respiratory tract. In addition there is considerable depression, a sensation of chilliness and often high fever. After a few days these symptoms pass off and the worker is apparently immunized for the remainder of the sea-

son. The symptoms in this instance are probably due to the inhalation of pollen, especially that of rag-weed. Landis (28) and Bowen (29) have also directed attention to the symptoms arising in threshers. The symptoms, they state, are often severe and alarming. They consist of a chill, vomiting, a flushed face, delirium, and a temperature which may reach 104°F. There is marked congestion of the mucous membrane of the respiratory tract and a copious nasal and bronchial exudate which contains quantities of microscopic dust particles.

In this category may be placed those instances of respiratory infections due to the inhalation of grain dust. While this dust contains much organic material it also undoubtedly contains considerable inorganic matter and is therefore to be regarded as a mixed dust. Formerly when the grain was transferred from vessels and cars to the elevator, and vice versa, the work was done by shoveling. Thomas F. Rochester (30) called attention to the fact that the "scoopers" or "shovelers" were extremely susceptible to acute respiratory disorders. Beginners often developed marked nasal irritation or a bronchitis, subacute in character. After two or three years an unusual type of broncho-pneumonia was of frequent occurrence. Because of the character of the employment it was commonly referred to in Buffalo as "elevator pneumonia." The attack was usually severe and at autopsy the lungs presented an engorged appearance at the bases and occasionally deep blood-red infarctions were found. The bronchial mucous membrane was without exception swollen and thickened. Convalescence in these cases was usually prolonged, being rarely under three months. A change of occupation was usually followed by complete restoration of health. The introduction of machinery for transferring the grain has apparently done away with the trouble.

Ramazzini, in 1670, in writing of the dust exposure in grain handlers, stated that: "The throat, the lungs, and the eyes sustain no small damage by it, for it stuffs and dries up the throat; it lines the pulmonary vessels with a dusty matter that causes a dry and obstinate cough." The acute symptoms in these cases are in all probability to be attributed to protein intoxication. In those who develop the evidences of organic changes in the lungs the condition can, without much doubt, be attributed to the large quantities of inorganic dust which is mixed with the grain.

Occasionally one of the acute infectious diseases is acquired by the inhalation of dust containing the specific organism. Rarely a fulminant and very fatal type of anthrax is encountered in which the disease is localized in the lungs.

The influence exerted by the trade process and the hygienic habits of the worker in his home are dealt with under the heading "The Relationship Between the Dusty Trades and Tuberculosis."

The Relationship Between the Dusty Trades and Tuberculosis

The most serious indictment against the dusty trades is that they cause, or at least predispose to, a high death rate from pulmonary tuberculosis. At first sight the evidence seems conclusive, but on closer analysis it is a question as to whether the facts as presented are altogether acceptable.

In considering the relation between the dusts and tuberculosis it is necessary to consider the inorganic and the organic type separately.

Inorganic Dust

Inorganic dust does produce definite pathological changes in the lungs. Of this there is no question. As has already been pointed out the changes may be very severe as in the case of silica, or they practically may be negligible, as in the case of soot. Because changes are produced by inorganic dust, the majority of writers on the subject assume that these changes render the individual more susceptible to tuberculosis. When we come to study the pathology of pneumoconiosis and tuberculosis, however, it is difficult to accept this view. The essential feature of the changes in pneumoconiosis is the formation of fibroid tissue. The arrest of a tuberculosis process, on the other hand, is brought about by the formation of fibroid tissue which walls off and prevents the spread of the tubercles.

In pulmonary tuberculosis the rapidity or the slowness of the spread of the disease is largely dependent on one factor,

namely, the formation of fibroid tissue. In the acute forms of tuberculosis one of the striking features of the pathological picture is the absence of fibroid tissue. Caseous areas coalesce, break down, and form ragged, ill-defined cavities with apparently no attempt on the part of the cells to limit the spread of the disease. On the other hand, in the slowly progressing form of chronic ulcerative tuberculosis, and especially in the so-called fibroid phthisis, there is always an overgrowth of fibroid tissue. If cavities are present there is a well-defined fibrous wall, and scattered throughout the invaded portion of the lung the evidence of fibroid tissue is plainly apparent.

In the section on "Pathology" reference has been made to the presence, in silicotic lungs, of areas of dense fibrous tissue which sometimes show the presence of tubercle bacilli histologically, or if emulsified and injected into guinea pigs, tuberculosis sometimes develops. This, I take it, is an evidence of extreme resistance on the part of the lung rather than susceptibility to the infection. It has furthermore been pointed out that when tuberculosis develops in the early stage of silicosis that the tuberculous foci hasten the formation of fibrous tissue in their vicinity and that the silicosis or fibrosis in these areas is far in advance of that in other portions of the lung.

Beattie (31), in considering the occurrence of tuberculosis among grinders, feels that dust does its damage only in "reducing the area of effective lung tissue, and probably therefore encouraging the lodgment of tubercle bacilli, but no one who has studied the pathological processes in the lung in cases of tuberculosis can be anything but skeptical when he is told that fibrosis encourages the spread of the tuberculous process."

Under the circumstances it is difficult to understand how a process which, in one case retards the activities of the tubercle bacillus, can under other circumstances render the condition for its spread more favorable. Wainwright and Nichols (32), in their study of anthracosis, expressed the belief that coal miners were less susceptible to tuberculosis than other individuals because of the diffuse fibroid changes in their lungs. That the carbon dust was in any way germicidal they did not believe. Klotz (17) and Smart (33) have expressed the same opinion.

In the older literature on tuberculosis there are not a few references on the value of lime dust in the treatment of tuberculous individuals. In the past decade Fisac (34) has, on several occasions, drawn attention to the immunity against tuberculosis displayed by persons whose occupation brings them into close contact with lime and plaster. It is his belief that each individual soil has its special chemical composition. This chemical composition will decide whether tubercle bacilli can get a foothold and multiply or whether the tissues react with a fibrous growth, checking the proliferation of the germs.

Another stumbling block in the case of inorganic dust is that it leads to changes in the lungs which are not to be distinguished from tuberculosis either by the symptoms or the physical signs; and even the roentgenogram may, at times, be difficult to interpret. My own experience with potters under my care at the White Haven Sanatorium is that a diagnosis of tuberculosis is often made when in reality the individual is suffering from a diffuse fibrosis of the lungs, associated with dilatation of the bronchi. The only certain means of distinguishing between the two conditions is the examination of the sputum. It is not safe to assume that because an individual in a dusty trade has the symptoms and physical signs of pulmonary tuberculosis that he is necessarily tuberculous. The strongest evidence in support of the frequency of tuberculosis due to or associated with inorganic dust are mortality statistics. Unfortunately much of the value of this data is vitiated by careless diagnoses. Far too often a diagnosis of tuberculosis is made without a sputum examination.

Furthermore, I now have knowledge of several potters who became tuberculous, had their disease arrested, and returned to their former work. In each case a number of years have elapsed and the patient has remained well in spite of being exposed to the same conditions as existed prior to his illness.

It is not to be understood that tuberculosis cannot develop in the presence of an existing fibrosis of the lungs. It undoubtedly does. But when it occurs, and this is true of potters, it commonly runs a latent

and more prolonged course than is the case of uncomplicated phthisis.

It has long been recognized that the age period at which potters succumb to tuberculosis is far beyond the average, and in addition the disease seems to be much less severe than the same amount of damage produces in other classes of patients. Most of the available statistics on the subject support this view. Thus the English mortality statistics for the three years ending 1902 (35) state that the mortality of potters, between the ages of 20 and 35 years, falls below that of occupied and retired males generally. At every other period, however, it shows an excess which amounts to no less than 74 per cent. at ages 45 to 55 years, and to 66 per cent. at ages 55 to 65 years. The principal excess falls under the head of respiratory diseases.

The following table (36) shows the proportionate mortality from consumption among potters, compared with that of males in the registration area in the United States:

Age at Death	Deaths of Potters, 1897 to 1906, from		Per Cent of Deaths Due to Tuberculosis Among	
	All Causes	Pulmonary Tuberculosis	Potters	Males in Registration Area, 1900-1906
15 to 24 years	46	11	23.9	27.8
25 to 34 years	68	36	52.9	31.3
35 to 44 years	84	37	44.0	23.6
45 to 54 years	78	22	28.2	15.0
55 to 64 years	72	14	19.4	8.1
65 years or over	36	7	19.4	2.7
Total	384	127	31.3	18.0

My own belief is that the higher incidence of tuberculosis among those exposed to inorganic dust is due to the fact that the dust acts as a convenient carrier of tubercle bacilli. In other words, it is not the preliminary injury which the dust is responsible for that predisposes the individual to tuberculosis, but the readiness with which tubercle bacilli may be carried into the respiratory tract by a dust-laden atmosphere. If an infected worker is careless in spitting on the floor, the sputum becomes dry and mixes with the dust, thus making it easier for the bacilli to reach the lung. Moreover, tubercle bacilli which become mixed with dust are, to a certain extent, protected and not as much exposed to the germicidal action of air and light as are those deposited in places free from dust.

In a survey made of the conditions in the gold mines in the Transvaal, South Africa, Watkins-Pitchford (37) found that 15.2 per cent. of samples of sputa obtained underground, chiefly from shafts and main traveling ways, showed the presence of tubercle bacilli, as against 2.5 per cent. of sputa collected in the living rooms or places of resort of white and native mine workers on the surface. The difference in these figures is probably to be ascribed to the fact that the dark, humid atmosphere of the mine aids in prolonging the life of the bacillus. It was also observed that the tubercle bacillus will survive in acid mine waters for a period of at least two months. If, therefore, a number of tuberculous infected miners are careless in spitting about the working places in the mine, the chance of infecting their fellow-workmen is great. And this danger of infection is quite independent of whether the workers have developed silicosis or not.

Beattie (31) has expressed the belief that the prevalence of tuberculosis among grinders is due to spitting on the wheels and hands, and thus contaminating the water which all use for wet grinding.

In a preliminary inquiry into the prevalence of pulmonary tuberculosis among the native mine workers in the Rand, Watkins-Pitchford, Orenstein and Steuart (38) state that tuberculosis is the main incapacitating factor in silicosis and that if open communicable cases of tuberculosis are rigidly excluded from the mines, the danger is greatly reduced.

Another factor to be considered is the exposure to infection entirely independ-

ently of the occupation. Tuberculosis has long been recognized as a house disease, for it is here that exposure occurs more often and more intensely than anywhere else. In an intensive study of 743 garment makers made by Reed and myself (39), tuberculosis in other members of the family was encountered in 27, or 6.7 per cent. of 402 males, and 13, or 3.8 per cent. of 341 females. The question of whether the worker is exposed to the disease in the home or has a family history of tuberculosis is extremely important. When such is the case it would be difficult to fasten the blame on the occupation. During the year 1913 there were treated in the Phipps Institute Dispensary 685 cases with a positive diagnosis of tuberculosis, and of this number ninety-six, or 14 per cent. were garment-workers. Twenty-seven, or 28 per cent., of the ninety-six, gave a family history of tuberculosis. Furthermore, the social histories of patients with tuberculosis but following an occupation other than that of garment making, showed that in 108 instances one or more members of the family were employed as garment-makers. It is thus seen that the worker is often exposed to danger of infection in the home.

In the present investigation the family histories of thirty-nine potters showed that the mother or a brother or a sister had died of tuberculosis in six instances (15.4 per cent.). In four more it was stated that the father had died of "potter's asthma," which might or might not have been tuberculosis.

Lanza (40) has emphasized the poor and often wretched conditions under which many of the zinc miners live. Unhygienic living conditions, carelessness of the miners themselves, and overcrowding in small three and four-room shacks are of frequent occurrence. In common with the South African observers, he found little tuberculous infection among the women which could be attributed to the men. Lanza believes, however, that this danger has been underestimated, although the condition may be explainable by reason of the short period during which many cases of miners' consumption emit tubercle bacilli before death. The danger to the children on the other hand is, he believes, a serious one. He concludes that aside from the hygienic supervision of working conditions underground, education of the miner against the spread of infection and supervision of miners' children, especially those of consumptive parents, are matters of vital importance.

In this connection it is interesting to note that in the state of Victoria, Australia, statistics show that while the incidence of tuberculosis is excessive among males in the Bendigo mining district, it is also excessive among the females who are in no way involved in mining operations. A careful study of sixty-five miners showed that 50 per cent. gave a clear history of close contact with cases of tuberculosis (41).

In closing this subject, I would redirect attention to Cornet's (42) original observations on the transmission of tuberculosis, namely, *The Diffusion of Tubercle Bacilli Exterior to the Body* and *The Mortality of the Nursing Orders.* An excellent review of both papers will be found in Tyndall's *New Fragments,* Appleton & Co., 1897. It has seemed to me, and the conviction grows stronger the more I study the problem, that the universal tendency is to emphasize the danger of dust and to minimize the fact that without the tubercle bacillus tuberculosis cannot be produced. Whether intentional or not, one gets the impression in reading of the dangers of dust that it is the dust which produces tuberculosis.

In a few instances conditions simulating pulmonary tuberculosis can be produced by dust, but in the overwhelming majority of cases no such change occurs. In other words we can eliminate dust, at least in the industrial sense, and still be exposed to the danger of tuberculosis if open active cases expectorate freely about the working places. Cornet showed from his study of the Catholic Nursing Orders in Germany that the mortality from tuberculosis was appallingly high and was to be ascribed to the close confinement of the nurses and to carelessness on the part of patients at a time when the danger of tuberculous sputum was but little appreciated. Let us keep before us the fact that some of the inorganic dusts are, *per se,* capable of producing serious pathological changes in the lungs; that others lead to slight changes; and that many of them produce no appreciable alterations whatever. Tuberculosis may be grafted on

such a process, but in such an event the exciting cause is the tubercle bacillus which has been thrown off by a pre-existing case of tuberculosis. In this connection may be quoted a paragraph from the second report of the British Royal Commission on Metalliferous Mines and Quarries (1914): "One conclusion at which we have arrived is that the cause of miners' phthisis is the inhalation of dust of crystalline silica, upon which is superimposed tubercular infection. Moreover, we agree that it is the tubercle bacillus which causes destruction of the lung and that dust is not necessarily dangerous to life." Professor Beattie said "The dust is not nearly as dangerous to life as the tubercle bacillus. If you get rid of the bacillus of tuberculosis the dust is not so bad."

Organic Dust

In the case of organic dust the problem is decidedly different. In the first place we can dismiss at once the question of injury to the respiratory tract as the result of the inhalation of organic material. The only factor in common is the part played by the dust particles as carriers of tubercle bacilli.

There is no doubt that among those who are employed in trades in which there is an exposure to organic dust there is often a high death rate from pulmonary tuberculosis. That organic dust *per se* is responsible, in that it produces pathological changes in the lungs, and thus renders them more susceptible to tuberculous infection, cannot be demonstrated. There is no pathological proof that such is the case and our experience with the X-rays is distinctly adverse to such an opinion.

In the case of inorganic dust the proof is indubitable and if undue exposure to this type of dust occurs, pathological changes are seen to result no matter what the habits or circumstances of the individual may be. Among those exposed to organic dust there are a number of factors to be considered in determining the high incidence of tuberculosis in this class of workers.

In the first place a large proportion of workers exposed to inorganic dust such as potters, miners, metal workers, are skilled and highly paid artisans while those employed as cotton mill operators, textile workers, etc., are, for the most part, unskilled and poorly paid. The relationship which exists between wages and ill-health is one of the most debated questions of the day. To what extent higher wages would do away with disease is problematical but there can be no doubt that a low wage often compels the worker to live in localities, the sanitary conditions of which are often poor. Furthermore, he is only too frequently undernourished, either because of an inferior quality of food or insufficient food. In this connection, however, it is only fair to state that the undernourished condition of many of the laboring class is to be ascribed to ignorance as to food values. In an intensive study made of the dietary of dispensary patients, many of them employed in the textile trades, Craig and I (43), found that they were getting only about four-fifths the quantity of food they should. This was due in part to an inadequate wage and in part to ignorance of food values. Formerly the working environment was often unsanitary, but in recent years tremendous strides have been made in the direction of improving these conditions.

In nearly all of the occupations in which the worker is exposed to organic dust it is well to remember that in the badly ventilated state of one factory compared with another, the home life and surroundings of the work people, poverty, heredity, age and sex are to be found conditions that favor the production of ill-health, and are therefore not to be ignored. This can best be illustrated by a few concrete instances. It is well known that the mortality from tuberculosis among tobacco workers is excessively high. This trade is usually listed as one of the dusty trades. Two years ago there were admitted to the White Haven Sanatorium, White Haven, Pa., a number of patients from one of the counties in an adjoining state. Among this number were ten young women who gave as their occupation that of cigar maker. All were suffering from a moderately advanced stage of tuberculosis. These women were foreign born, mentally stupid, untidy and of the type that made it reasonably certain that their living conditions were far from being good. They all came from a group of small manufacturing towns closely adjoining each other. These towns are built on a marsh, the

streets are, as a rule, unpaved, underdrainage is lacking, and as a personal inspection showed, the living conditions were vile. There was but one visiting nurse, and she informed me that the enforcement of cleanliness among these people was almost impossible. In addition to their ignorance of hygiene a low wage forced them into the meanest quarters and in addition rendered an adequate diet impossible. That is one side of the picture.

The other side consists of the cigar factory in which these women were employed. It was modern, well lighted, well ventilated and the reverse of being overcrowded by workers. As I had never personally inspected a cigar factory I accepted the dictum that this trade was a dusty one. There was little dust of any kind and so far as tobacco dust is concerned there was none at all for the following reason. In as much as the tobacco leaves are very fragile and must be handled and manipulated constantly in manufacturing the cigar, it is apparent that this can be done only by keeping them moist and pliable. How quickly a dried out cigar can be crumbled into dust is familiar to all. Tobacco dust is absent from this industry, therefore, because of economic reasons.

Dr. E. H. Funk who inspected several large cigar factories for me noted the absence of tobacco dust. Smith and Miller (44), in a hygienic survey of forty-eight cigar manufacturing establishments in Philadelphia concluded that cigar making is not necessarily a dangerous industry. Certainly so far as tobacco dust is concerned it is not. In common with many other manufacturing plants, many cigar making establishments contain varying amounts of mixed dust which is carried in on the shoes of the workers or sifts in through the windows. This is often allowed to accumulate for months at a time. Among the cigar makers whom we studied roentgenologically, none of them showed changes in the lungs which differed in any way from those of the city dweller and in some instances they had followed the trade for twenty-five years or more.

Another fallacy regarding the possible effect that dust may have on ill-health is the failure of our statistics to separate the various occupations into trade processes.

A man employed in a felt hat factory, for instance, will almost invariably designate his employment as a "hatter." It is to be borne in mind, however, that there is a wide difference in the character of the different processes concerned in the manufacture of a hat. Those handling the raw fur which composes the felt may, if exhausts are not employed, be exposed to dust. On the other hand, those who mold the felt into form are as far removed from dust as possible as they work in a steam saturated atmosphere. While this industry is often included among the dusty trades it is noteworthy that of the hatters we have seen at the Phipps Institute, all have been from the molding department. If tuberculosis is unduly prevalent among the workers in this process it is to be ascribed to catarrhal changes induced by exposure to sudden changes in the temperature and to the dampness. Dust certainly plays a very small part.

The cause and effect in the case of inorganic dust is direct and apparent. In the case of organic dust, however, there are a number of contributing factors to be considered and unless we have a complete picture of the worker's life it is unsafe to ascribe all the ills to which the worker may be heir, to the nature of his occupation.

This was well brought out in an intensive study of 743 garment workers made by Reed and myself (39). In this study it was shown that among those workers who took their meals irregularly or who had insufficient or inadequate food and among those who did not get sufficient sleep the incidence of tuberculosis was much higher than in the case of those leading a regular life and obtaining sufficient nourishment. Among the women who were getting insufficient food and sleep the incidence of tuberculosis was three times that of those of whom the reverse was true.

As in the case of inorganic dust, the undue incidence of tuberculosis in association with organic dust is to be explained by the presence of infected individuals who are careless in expectorating on the floors of workrooms and thus permitting the tubercle bacilli to become mixed with the dust.

PATHOLOGY

Inorganic Dust

Before taking up the successive changes which are produced by the inhalation of dust, attention should be directed to the fact that the air passages are provided with a natural defensive mechanism against the entrance of foreign material. The average amount of air which a healthy grown person takes in at each breath has been estimated to be about one-half a litre (about 30 cubic inches). Now there is being little air breathed by the great mass of people, and especially by city dwellers, which does not contain particles of dust and bacteria of various sorts. In an analysis of the outdoor air from various parts of New York City, Prudden (45), found that the average number of bacteria in 10 litres to be 376, and of moulds six.

In buildings and in private houses the number of dust particles and bacteria is greatly reduced. It has long been recognized that the solid particles which we breathe in with the air either through the nose or mouth do not come out with the expired air but are retained on the moist surface upon which the air impinges, going in and coming out. Straus in one experiment found that the expired air contained only forty bacteria, whereas the inspired air held over twenty thousand. Not only bacteria but dust particles are caught largely in the nose, or mouth or upper pharynx. The nose especially is a very efficient dust filter and many dust particles are held on the moist surface of its mucous membrane. When the air current leaves the nose it turns downwards and impinges against the posterior wall of the pharynx. Much of the dust is arrested at this point. The mouth is not so well adapted as the nose for moistening the air and removing the dust particles and bacteria. For this reason those who suffer from nasal obstruction or who breath through the mouth from other causes are more susceptible to "colds" than those who breathe through the nose. A very considerable proportion of the dust in the inbreathed air is spat out, swallowed or removed from the nose.

According to the experience of Watt, Irvine, Johnson and Steuart (25), silicosis is more apt to develop in a mouth breather than in a nose breather. In our own investigation we were at a loss to explain the variations noted in those exposed to the same kind of dust and for an equal length of time. This difference in resistance of individuals to dust was also noted by Arlidge (46), many years ago. Some workers will show in their lungs marked pathological changes while others will show very little evidence of damage, although the circumstances are identical in both. Thinking that variations in the upper respiratory tract might explain the matter, Dr. George Fetterolf, at our request, examined the nose and throat of a number of cement workers. He was unable to find anything, however, which threw any light on the matter. There can be no doubt, however, that the mechanical defense in the nose is superior to that of the mouth and that mouth breathers are therefore more susceptible. On the other hand, there seems to be no doubt that individuals vary almost as much in their reaction to inert dust particles as they do to bacteria. For instance, a potter whom we have had under observation was exposed to clay and flint dust for nearly forty years and yet his lungs showed but a moderate grade of pneumoconiosis. On the other hand, many of his fellow workmen equally exposed and in some instances less so, showed extreme fibroid changes.

Let us return, however, to the dust particles. The floating material which is carried past the upper defenses enters the trachea and bronchi and lodges in their moist walls. Nowhere in the body has nature provided a more ingenuous arrangement for expelling unwelcome intruders. Completely lining the larger air tubes like a mosaic, are myriads of tiny cells shaped something like a narrow short club and set upon end side by side. Projecting from the free ends of each one of these cells is a number of minute hairs. These cells are called cilia. "These myriads of cilia, year in and year out, day and night, while life lasts, are constantly swinging their free ends back and forth, bending as they recover, and then with a quick snap forward so that any small object which lodges on the walls of the larger air tubes—since all the cilia act in rhythm—is swept upwards toward the mouth, away from the perilously delicate and

sensitive lungs." (Prudden).

By the action of the cilia the bulk of the dust particles is arrested and removed under ordinary conditions.

The particles which escape the cilia and penetrate deeper into the respiratory tract encounter another defensive force, namely, the phagocytes and the large cells lining the alveoli. These alveolar cells have been termed the normal "respiratory scavengers." Some of the dust particles are taken up and retained by the large cells lining the alveoli; others are seized by the phagocytes. In the case of bacteria the phagocytes destroy them by absorbing and digesting them. In the case of inert dust particles they are either expelled in the secretions or carried to some point where they will do the least damage. But even these fail to deal with all the dust particles and when overwhelmed as in great exposure, the particles penetrate the mucosa and reach the lymph spaces.

The last defense is the lymphatic system of the lungs and pleura. The lymphatic system of the lungs consists of a series of irregular, narrow channels which open into larger and still larger trunks. The terminus of this system is formed by the bronchial lymph nodes. An interesting observation regarding the direction of the lymph flow in the lungs has been made by Dunham (47). By means of large serial sections he has shown the presence, in the lymph vessels, of valves pointing definitely toward the pleura. These lymphatic vessels have their origin in some instances, in the interalveolar spaces and accompany the large vascular trunks and the bronchi toward the root of the lung; in other instances, they originate in the sub-pleural spaces and communicate on one side with the pleural cavity and on the other anastomose with the lobular and interlobular lymphatics. Many dust particles are taken up by these vessels and deposited in the lymphatic filters, the lymph nodes.

In spite of these defenses, however, there accumulates in the pulmonary tissues a considerable amount of foreign material. The amount will depend almost entirely on the degree of exposure. In all urban dwellers who reach adult life there is some evidence of dust accumulation in the lungs. At birth the lungs are of a pink color but as time goes on and the dust particles gradually accumulate, principally in the form of carbonaceous material, the lungs assume the familiar greyish color seen at the autopsy table. Pneumoconiosis of this degree produces but slight organic changes and rarely interferes with health.

While all forms of inorganic dust bring about essentially the same change in the respiratory tract, they vary one from another, in the extent of the lesions produced and the severity of the symptoms. The sharper and more angular the dust particles, the greater will be the amount of mechanical injury and hence the greater the inflammatory reaction. The most dangerous of the inorganic dusts is the siliceous.

If exposure to the dust is sufficiently long there develops a pharyngitis and later a bronchitis. Between simple irritation as the result of brief exposure, and extensive tissue changes, as the result of prolonged exposure, varying degrees of pathological alteration may occur. It is noteworthy that the changes are very slow in their evolution and except under extraordinary circumstances, it takes years to bring about serious damage.

In the case of a worker exposed to unusual quantities of inorganic dust the majority of the particles are, for a varying length of time, arrested by the moist surface of the mucous membrane, by the action of the ciliated epithelium and by the phagocytes. Sooner or later, however, these defensive forces weaken and finally the dust passes into the lymph channels and also along the finer bronchi until it reaches the parenchyma of the lungs. As a foreign substance it then sets up a chronic inflammatory process.

Our experience is in agreement with that of Arlidge (48) and Watt, Irvine, Johnson and Steuart (25) in that the anterior and inferior marginal portions of the lungs, where expansion is most free, are always less affected than the posterior and apical portions. Radiographic evidence shows also that the change is frequently slightly more advanced in the right lung than in the left.

The microscopic changes which occur in pneumoconiosis have been described by Wainwright and Nichols (32), Haythorn (49), Klotz (17), and by Watt, Irvine, Johnson and Steuart (25). The

evolution of pneumoconiosis is characterized by several well defined stages:

(1) The very earliest changes have been well described by Wainwright and Nichols who, because of accidental deaths, were able to study the lungs of men who had worked in a coal mine but a few months. In these cases they found that some of the epithelial cells lining the alveoli had become swollen and contained particles of coal dust. Sometimes a few larger desquamated cells containing much pigment are found in the alveoli lying loose with some detritus and free dust particles. Even in this early stage, dust particles are seen in the walls of the air vesicles and around the small bronchi. In this stage there is no evidence of connective tissue proliferation. In the case of silicosis, however, it is likely that fibrosis begins at an earlier period than is the case in anthracosis owing to the more irritating properties of the silicious dust.

(2) In this stage it is evident that the dust particles have penetrated all of the first defenses and have gained entrance to the lymphatic channels. The swollen cells containing dust particles are much more numerous and in addition there are present large mononuclear phagocytes filled with dust particles. They lodge first mainly in the small aggregations of lymphoid tissue which lie between the alveoli and along the course of the perivascular, peribronchial and interlobular lymphatics. In a microscopic study of seven silicotic lungs, Watkins-Pitchford (24) was able to demonstrate the situation, outline and dimensions of the mineral particles by means of polarized light. Watt, Irvine, Johnson and Steuart (25) describe the stage as one of *discrete fine fibrosis* which results from the aggregation of dust particles along the course of the small blood vessels and bronchioles and to a limited extent in the interalveolar, interlobular, and sub-pleural tissues. The fibrosis at this stage is almost entirely perivascular and peribronchial. The alveolar walls generally are not thickened and the alveoli are not reduced in size, although they show a certain amount of catarrh.

(3) In this stage the accumulations of dust particles gradually increase. The early perivascular and peribronchial fibrosis becomes more accentuated and in the interalveolar, interlobar and sub-pleural tissues become thickened. In addition nodular collections appear at the points of junction of the various lymph channels where small receptaculi are formed. These nodules become so prominent that they are readily felt by the finger and at times the course of the lymph channel can be detected by the sensation of touch. At the points where the dust collects the fibrous tissue is arranged in concentric layers, and toward the periphery the dust particles are especially numerous. The fibrous masses are well seen in the septa that run in from the pleura. In places considerable sized black triangles with their bases on the pleura are seen, showing where the septum has been completely filled up with dust. The striking point in the whole picture is the extensive plugging and obliteration of the small and medium-sized lymphatics and the compression of the large ones. In the pleura and the peribronchial lymph nodes the picture is a similar one (Haythorn). In this stage the bronchial and mediastinal lymph nodes become enlarged. By some, the plugging of the lymphatic vessels is considered as predisposing the individual to lobar pneumonia. Klotz leans to this view as the result of his studies. Although pneumonia prevails to a considerable extent among the "blacks" in the Rand mines, those who have studied the subject state that silicosis seems to exert little influence. The new arrivals seem especially prone to develop pneumonia but after a residence of six months the incidence of this disease is no greater among the black than among the white miners.

(4) The final and advanced stage is characterized by a general nodular dissemination throughout both lungs. In many places the nodules coalesce forming large fibrous areas. These advanced changes take place as a rule only after years of exposure.

(5) A most important change, from the view point of prevention, was noted by Wainwright and Nichols. They found that in the case of individuals who had formerly been coal miners, but who for many years had not followed that occupation, the lungs no longer showed signs of irritation. The swollen epithelium had subsided and again become normal, and neither the cells nor the alveoli contained

dust. The deposits of dust in the alveolar walls, the septa and perjbronchial tissue, however, still remained, as did also the connective tissue thickening.

The changes described above as occurring in man have also been produced in animals. Beattie in an experimental study with guinea pigs drew the following conclusions:

(1) That the effects produced in the lungs of guinea pigs which have been exposed to the inhalation of dust correspond with those produced in man. (2) That the effects produced vary with the length of exposure and particularly with the quantity and quality of the dust which is inhaled. (3) The more concentrated the dust in the atmosphere the more rapidly will fibrosis be produced and the more irritating the dust the more intense will be the fibrosis. (4) The inhalation of such stone dusts as are rich in silica seems to be particularly injurious, and (5) that the powdered shale used in these experiments does not produce more irritation in the lungs than does coal dust, and judging by the comparatively non-injurious effects of coal dust and the general health of the workers, powdered shale may also be regarded as comparatively harmless.

In a study of the lungs of rats exposed to dust in gold mines in the Rand, Johnson (25) states that the development of silicosis in these animals very closely resembles that which occurs in man. The condition is definitely progressive and can be subdivided into stages similar to those noted in miners.

Gross Anatomy

The color of the lung which contains dust pigment will vary according to the extent of the pneumoconiosis and the character of the dust. It may be of varying shades of gray, intensely black (anthracosis) or it may be red, as in the cases of siderosis due to the red oxide of iron reported by Zenker. The slate gray color of the lungs of the city dweller is familiar to all. This constitutes the early change in pneumoconiosis. In the absence of any other lesion such lungs are regarded as normal. Even in the stage of discrete fine fibrosis the lung shows no gross change although occasionally "precocious" macroscopic nodules are noted and the smaller vessels and bronchioles may be slightly raised above the cut surface and definitely palpable.

When the fibrosis reaches a more advanced stage the mottling on the surface of the lung is quite distinct and the interlobular septa are readily distinguished. Occasionally the surface of the lung is scarred and distorted. On palpating such a lung it is aerated throughout but is distinctly less elastic than normal. The walls of the small blood vessels and bronchi are seen to be thickened and they stand out distinctly on the cut surface. Pigmented areas are also seen and it is common to find a number of small microscopic fibrous nodules under the pleura or in the substance of the lung. The bronchial lymph nodes are fibroid and deeply pigmented.

In the advanced stage the lung is hard and more or less solid as the result of fibrosis and is always heavier than normal. In a series of silicotic lungs Watkins-Pitchford (50) found that they weighed from two to three times that of the normal organ. This is due to some extent to the increase in the amount of fibrous tissue but largely to the deposition of dust. In those exposed to siliceous dust Kussmaul (51), found that after incinerating the lung the residual ash might constitute 40 or 50 per cent. of the total. Arlidge (52), in an analysis of the ash from a potter's lung found it contained 47.78 per cent. of silica.

The dominant feature is the fibrosis. The organ often cuts with some difficulty and gives a creaking sound owing to the presence of fibrous nodules. Grittiness, which is so often alluded to, is, in the experience of Watkins-Pitchford rarely present even in an advanced stage of silicosis. The cut surface feels rough and gritty, partly as a result of the thickened vessels and bronchi, and partly as the result of the deposit of mineral material. Massive fibrous areas may be seen. In some instances they are due to the coalescence of smaller areas, in others they are due to a chronic tuberculous invasion of the lung. In many instances the latter show no gross evidence of tuberculosis, but careful histological studies will often show a few tubercle bacilli and emulsions made from this tissue, if injected into a guinea pig, will often produce tuberculosis. Clinically it is quite likely that such lesions are so latent that they are innocuous. In cases of silicosis more than in any other form of pneumoconiosis the acute forms of tuberculosis, acute pneumonic phthisis and tuberculous broncho-pneumonia, are apt to develop. It has also been noted that when a tuberculous focus occurs in a silicotic lung it seems to hasten the advance-

ment of the silicosis and that about such an area the condition presents itself as a far advanced stage of silicosis while elsewhere the lung may appear to be only in the first stage of the disease. The dense fibrous areas containing a few tubercle bacilli probably represent a slight tuberculous focus which has been healed.

In advanced stages of pneumoconiosis the pleura is usually adherent to the chest wall, although the extent of the adhesions varies somewhat with the nature of the infecting dust. In anthracosis the adhesions may be very slight while in silicosis they are usually dense, the lung being adherent not only to the chest walls but to portions of the pericardium. In quartz miners Purdy (53) states that a common finding is an old or recent pleurisy; notably in the locality of the fifth and sixth ribs in the axillary region.

The only other condition likely to give rise to such extensive pleural thickening is chronic tuberculosis. In the case of the latter disease the lung is shrunken, ragged and collapsed which is in marked contrast to the full, firm and much weightier lung seen in pneumoconiosis.

In common with all forms of pulmonary fibrosis no matter what the exciting cause, dilatation of the bronchi is a common finding in the advanced stages of pneumoconiosis. Calvert Holland (1843) was the first to direct attention to this condition. The dilated bronchi are to be found, as a rule, in the portions of the lung which are most markedly fibroid, namely, about the roots and at the apices.

In common with other forms of chronic inflammation of the bronchi the walls may be thickened and hard. The mucous membrane may be of a dull red tint, or brownish in color, the discoloration being evenly distributed or occurring in patches. The membrane may be thickened in some places and in others thinner than normal. Very often the fine capillary vessels present a varicose appearance.

Emphysema located along the anterior margin and at the bases of the lungs is of frequent occurrence. Emphysema may be noted also between the areas of fibrous tissue.

Cavity formation, the result of tissue necrosis is not common in uncomplicated pneumoconiosis, although it does occasionally occur. Among zinc miners Lanza (40) states that necrosis as the result of pyogenic infection is not infrequent. This manifests itself clinically by the breaking down of the lung tissue and the expectoration of characteristic slaty blue sputum. Abscess formation in the lungs has also been noted among zinc miners. When a cavity is present it is to be regarded as being tuberculous in character until proved otherwise.

A not infrequent finding in anthracotic lungs is a pseudo-cavity due to a saccular dilatation of a bronchus. It is frequently located at the apex, and is readily distinguished as being of bronchial origin by the smooth wall which is continuous with that of the bronchus. Clinically it is important to keep in mind the fact that the bronchi are often dilated in the apical portion of the lung as the physical signs are not to be distinguished from those obtained in the case of a true cavity.

The diaphragm shows most interesting changes. In advanced cases it is nearly always distorted and its functional capacity greatly reduced. As fibrous changes first occur at the root of the lung and often extend downward, the phreno-pericardial angle is often obliterated comparatively early in the disease. This causes a restriction of the inner half of the leaflet usually most marked on the right side. In the advanced stage a sharp angulation of one or both leaflets is often seen, especially on inspiration. In many of these cases it is easy to demonstrate the presence of a fibrous strand extending from the hilus of the lung to the diaphragm at the angular point. This evidently anchors the diaphragm at this point and causes the leaflet to become peaked or angular on inspiration.

If the fibrosis is more decided on one side than on the other, the most affected side will show a greater amount of retraction and the heart will be displaced or drawn toward that side. In as much as pneumoconiosis generally produces a more or less uniform bilateral fibrosis, displacement of the heart is not a marked feature. In the terminal stages dilatation of the right heart is commonly present. It is caused by the obstruction in the pulmonary circulation as the result of the fibroid changes which have occurred in the lung.

Organic Dust: Morbid Anatomy

Although cotton fibers have been demonstrated frequently in the sputum of operatives in cotton mills the invasion of the lung by the material has not yet been shown. And this holds true for all forms of vegetable and animal fibre with the possible exception of particles of dust obtained from the abnormally hard woods.

References in the literature to pathological studies of the lungs of those who have been engaged in trades which have been associated with much dust of animal or vegetable source, are not of frequent occurrence. Or perhaps it would be more accurate to state that such studies rarely have been made with reference to the effects such dusts might have on the lungs. There can be no doubt that the lungs of such individuals have been studied but it is significant that but few references have been made to changes which are analogous to those encountered in the lungs of those exposed to inorganic dust.

Greenhow (20) for instance, studied several lungs obtained from flax dressers. These lungs grossly presented much the same appearance as those seen in potters or others engaged in work which causes exposure to inorganic dust. On incinerating them, however, it was found that the resulting ash was partially dissolved in boiling hydrochloric acid; the part left was dissolved by hydrofluoric acid, thus proving it to be silica.

Hirt (54), states that in two cases where the lungs of horse hair workers were indurated precisely like those of sandstone workers, he could find no trace of horse hair, and therefore concludes that the effect was probably due to the inorganic constituents of the dust.

In considering the effects of inorganic dust all the evidence points to the fact that even this type varies greatly in accordance with the size, hardness and regularity of the dust particles. In as much as all the vegetable and animal dusts with but few exceptions, are devoid of these characteristics it is difficult to understand how they could cause injury. When pathological changes are encountered in the lungs of those who have been exposed to organic dust the lesions are to be ascribed to inorganic material—in other words, it is a mixed dust.

Finally, it might be said that the roentgen rays reveal indubitable evidences of chronic pathological changes in the lungs of those exposed to inorganic dust; that these changes are definitely progressive and develop in a fixed sequence. On the other hand no such changes have been noted in the lungs of those exposed to organic material even when the exposure has been as long as thirty years.

SYMPTOMS

Inorganic Dust

Pathological changes due to an inert substance, such as inorganic dust, differ from those of bacterial origin in that they are slowly but definitely progressive so long as the exposure to the dust continues. Unlike the changes caused by bacteria there are no periods of quiescence and recrudescence. Furthermore, the changes are progressive only so long as the irritant is constantly operative; when the individual is no longer exposed the lesions become non-progressive, although those already present persist throughout life.

Exposure to any form of dust, even for a brief period of time, is apt to cause considerable irritation of the upper air passages, as manifested by a tickling sensation in the throat, cough and sometimes sneezing. After the worker becomes accustomed to the dust he may have no evidence of respiratory irritation for years. In other words, during the first stage of pneumoconiosis there may be no symptoms or only those indicative of a pharyngitis. Even in the early stages of the resulting fibrosis, symptoms may be slight or entirely wanting. This long period of latency is not to be wondered at in a condition whose pathological beginnings are fine and generalized. The symptoms will naturally be correspondingly insidious and their onset more elusive in character than is the case with most other pulmonary diseases. To this latency, in spite of definitely progressive lesions, is to be ascribed the difficulty of recognizing the condition by ordinary clinical methods. That the fibrosis is the principal cause of the respiratory symptoms there seems to be no doubt.

The more the effects of inorganic dust are studied the more apparent it becomes that silica is by far the most injurious

form encountered. When the worker is subjected to this type of dust in great concentration the fibroid changes in the lungs develop relatively quickly and evidences of respiratory damage are correspondingly early in their appearance. The evil effects of silicious dust have been well shown among the gold miners in South Africa and by Lanza (40) among the zinc miners in southwestern Missouri. In both instances the dust is almost entirely silicious in nature and, by reason of the work being done in closely confined underground chambers, the concentration is great. Under these circumstances early pulmonary changes may be noted in four or five years.

Even in the case of dusts which are not purely silicious the amount of damage seems to be proportionate to the amount of silica they contain. If silica is absent or exists in small amounts only, the evolution of the process is very slow and years may elapse without there being any appreciable damage. Thus among sixteen cement workers studied in the present investigation only one had a cough. The remainder were entirely free from respiratory symptoms. While the average length of time these men had been in the industry was comparatively short (average 10 years) the dust exposure was most intense. Among potters whom I have studied (55), serious symptoms rarely arose until the worker had been in the trade from ten to twenty years. In the present study freedom from any respiratory symptoms was noted after 38, 41, 49, 51, and 54 years of employment among thirty-nine potters. On the other hand, one man after six years in the trade had had an irritating cough for over a year.

The progress of the disease is, therefore, commonly slow but when induration of the pulmonary tissues has once been established, it tends to advance more rapidly, though insidiously. As a rule, *cough*, often unproductive in character, is the first symptom, especially in the milder types of pneumoconiosis. This is usually worse in the morning, is often paroxysmal, and tends to become increasingly severe. Following the morning paroxysm of coughing the worker frequently vomits. That eating seems to play some part in the vomiting is apparent by reason of the fact that the omission of breakfast minimizes or abolishes the paroxysm. By some the coughing paroxysm is ascribed to irritation of the gastro-intestinal tract as the result of the ingestion of the dust. Occasionally the cough may be wanting entirely. In the great majority of cases the dry, hacking cough is sooner or later associated with *sputum*. The expectorated matter at first is small in amount, tenacious and greyish in color as the result of small specks of carbonaceous matter. Even when the dust is not black in color, as in the case of clay or flint dust, the expectorated matter is usually greyish in color. In coal miners the expectorated matter is often inky black in color, hence the name "black-spit," sometimes applied to anthracosis. If the dust is composed of some distinctive coloring matter, such as red oxide or iron or ultra-marine blue, the sputum maybe either red or blue in color. Lanza (40) states that among zinc miners the sputum is often very characteristic, in fact diagnostic, being of a marked slaty blue color and of a very tenacious consistency. In doubtful cases sputum of this character is sufficient to determine a diagnosis. The color is due to the inhalation of dust which has become imbedded in the tissues. In this case the dust is composed of powdered chert which is an impure flinty rock, including the jaspers. In the stage of extreme fibrosis the sputum often becomes mucopurulent and is frequently blood streaked. Small *hemoptyses* are not uncommon. Of 433 cases of silicosis studied by Lanza (40), 33 per cent. of those in the first stage had hemorrhages; in the second stage, 10.5 per cent.; in the third stage, 20.5 per cent. of the uncomplicated cases and 30 per cent. of those with an added tuberculous infection. Night sweats are uncommon.

If there is an associated *bronchiectasis* located in the inferior portions of the lungs the sputum may be profuse and fetid in character. Dilatation of the bronchi at the apices, on the other hand, is not associated with excessive expectoration or fetor of the sputum as the drainage is good and stagnation of the bronchial secretions does not occur. In common with other individuals subject to chronic pulmonary lesions these patients are frequent sufferers from acute colds, and during such attacks the amount of sputum is markedly increased.

Shortness of breath on exertion or a more or less constant dyspnea sooner or later makes its appearance. In the case of true silicosis, dyspnea usually antedates the appearance of a cough and for a varying period of time may be the only symptom complained of. It is often more annoying than the cough and although in the milder cases it may be noticeable only on slight exertion, it is felt in the severer forms even when the patient is at rest. At times it has some resemblance to asthma, the breathing being wheezy in character. In such cases the inhalation of the dust seems to be the exciting factor. In a potter whom we had under observation the "asthmatic" symptoms entirely disappeared when he gave up the trade after being in it over forty years. The shortness of breath has been ascribed partly to the associated emphysema and partly to the replacement of pulmonary tissue by the fibrosis. A most important factor, in our opinion, is the functional capacity of the diaphragm. We found that in those cases in which both leaflets of the diaphragm were freely movable, shortness of breath was absent. This was so constantly the case that accordingly as the patient gave a history of being free from dyspnea or one indicating this symptom, we could predict the fluoroscopic findings in regard to the diaphragm. Those who have studied the effects of silica in the lungs of gold miners in South Africa state that shortness of breath on exertion is one of the earliest of the cardinal symptoms and is to be ascribed to the loss of pulmonary elasticity, as evidenced by diminished chest expansion (56).

As the disease advances a feeling of tightness and constriction of the chest is often present and in addition definite *pleuritic pain* may be complained of. In silicosis chest pain is often an early symptom and one that persists throughout the course of the disease although it may remit in the terminal stage. The location of the pain is variable but it is worth remembering that the pain or sense of tightness in the chest is often referred to the upper parts of the thorax—that is, in that portion where the early changes of pneumoconiosis first make their appearance.

The *general appearance* of the worker whose lungs show an early fibrosis is that of robust health. As the condition progresses, however, some impairment of the health may manifest itself. The individual reaction to the condition is, however, extremely variable. Some will show no appreciable deterioration in health in spite of marked fibroid changes, while others will begin to lose flesh, become anemic and even in uncomplicated cases, present a picture not unlike that of chronic tuberculosis. In the early stages the blood pressure is normal but as the process advances and the right heart dilates as the result of obstruction in the pulmonary circulation, the pressure is low.

Uncomplicated pneumoconiosis is often *afebrile,* or if fever is present it is very slight. The presence of any considerable amount of fever should arouse the suspicion that the condition is complicated by *tuberculosis.* The behavior of a tuberculous infection engrafted upon pneumoconiosis is subject to some difference of opinion. The teaching on this subject has, for many years, been to the effect that the clinical manifestations of the tuberculous disease were modified. This has certainly been my experience in the case of potters. Such patients do not look tuberculous; are afebrile, except in the terminal stage; and even then the temperature fluctuations are often not marked. South African observers, however, express the belief that with the occurrence of a tuberculous infection in those suffering from silicosis, the symptoms and signs are predominantly those of the former disease. Lanza, whose investigation was concerned with the same type of dust, namely silica, states that "even when tubercle bacilli are present in the sputum the lack of resemblance is plain and persists until the last few days' of life, when the patient being practically moribund, the resemblance to tuberculosis is more apparent."

It is to be borne in mind that the tuberculous infection, whether previously quiescent or due to a reinfection, is secondary in producing disability, though it may hasten death. While there are cases in which the tuberculous infection seems to exert but little influence on the general health of the patient, its tendency is to aggravate the symptoms and to cause a rapid decline in health. It is to be regarded in the majority of instances as a terminal infection.

Organic Dust

The inhalation of pure organic dust may temporarily cause some irritation of the upper respiratory tract and thus bring about coughing or sneezing or, if a foreign protein is inhaled, an asthmatic seizure may be induced. Other than these there are no symptoms as the type of dust is incapable, with rare exceptions, of causing structural changes in the lungs. The effects of organic dust have been fully discussed in the section on *etiology*.

PHYSICAL SIGNS

Inorganic Dust

In the great majority of cases nothing abnormal is to be detected in the so-called first or second stages of pneumoconiosis. This will be the case when the X-ray shows distinct evidences of changes, particularly in the second stage. Given the type of dust and the length of exposure one can hazard a shrewd guess as to the stage of the disease. When the disease enters the third stage, the physical signs are marked but not distinctive of the condition.

The physical findings in the third stage are as follows:

Inspection.—Expansion of the chest is usually deficient and may be more marked on one side than the other. There may be considerable retraction of one side of the chest and where this is present the heart will be drawn toward that side. Occasionally the chest is somewhat emphysematous in type. Clubbing of the fingers is commonly present and is indicative of an associated bronchiectasis.

Palpation.—This will aid in determining the degree of expansion on the two sides and the location of the apex beat of the heart.

Percussion.—If emphysema is present the note will be hyper-resonant. In the majority of instances, however, there are areas of dullness overlaying the dense fibrosis. The dullness is apt to be more marked in the mid-portion of the lungs and towards the apices. It may be greater on one side than the other. A tympanitic note may be elicited at the apex or near the angle of the scapula. When present it is an indication of bronchiectasis.

Auscultation.—The auscultatory signs are varied. The breath sounds may be diminished in intensity and broncho-vesicular in character. In many cases the breathing is rapid owing to involvement of the diaphragm. If the bronchi are dilated the breathing over the affected area is bronchial or cavernous; this may occur at the apex or near the angle of the scapula. In the latter area one can be almost certain that the signs are due to bronchiectasis while at the apex they may be due to this condition or to a cavity.

Râles of all descriptions may be heard all over both lungs. The condition may be mistaken for chronic bronchitis or owing to the wheezy character of the breathing and the presence of sibilant or sonorous rales, asthma may be suspected.

The voice sounds will vary—they may be suppressed in some areas and exaggerated in others; if the bronchi are dilated, both the spoken and whispered voice are readily transmitted at certain points, namely, the apex or about the angle of the scapula.

The heart shows nothing distinctive. In the advanced stage evidences of dilatation of the right heart may be present.

Organic Dust

As no lesions are produced in the lungs by organic dust physical signs are lacking except in those instances where an asthmatic seizure is induced by the inhalation of a protein containing dust.

X-RAY EXAMINATION

The study of this condition by means of the roentgen rays is the greatest advance which has yet been made. In the past few years several excellent reports of this nature have been published. During the past two years Pancoast, Miller and I (57) have studied a large number of individuals who have been exposed to dusts of various sorts.

In regard to the effects of *organic dust* the subject can be dismissed with the statement that these individuals show no more marked changes than are encountered in those who have dwelt all their lives in large cities. When the evidence of pneumoconiosis is slightly more marked than normal in these cases, it is to be ascribed to the fact that there is a considerable amount of inorganic material mixed with the organic dust. This was notably so in a number of carpet makers we examined.

The roentgenologic picture in the case of those exposed to *inorganic* dust is, in the great majority of cases, characteristic, except perhaps in the very early stage. As a rule, the changes as seen in the roentgenogram correspond closely to the pathological processes.

The first stage is characterized by an increase in the hilus shadows and a thickening of the usually prominent trunk shadows, and an undue prominence of the finer linear markings. It is the rule that the increase in thickness of the bronchial trunk shadows is fairly uniform, which is the main dependence in distinguishing the case from one of peribronchial tuberculosis, but there are exceptions which make the roentgenologic diagnosis difficult or uncertain. The descending trunk shadows seem to be more marked on the right than the left but the interference offered by the heart shadow on the left side may hide a similar condition there. Abnormalities of the diaphragm excursion occur in all stages, even the first. In the first and second stages, however, there is no general rule. The position of the excursion during ordinary respiration between the limits of extreme inspiration and expiration is very variable. The most common finding, in our experience, was a restriction at the inner portion of the right leaflet during full inspiration and either a decided flattening or a concavity of the outer aspect.

The second stage is characterized by a more or less uniformly arranged mottling throughout the lung structure due to the deposition of dust in the lymph spaces, cells and fibrous tissue interspaces, with the addition of a certain amount of localized fibrosis. This stage comprises what has usually been regarded as the typical case of pneumoconiosis. Its onset seems to depend largely upon the character of the dust inhaled. It occurs early in those exposed to silica, comparatively early in coal miners, certain metal grinders and somewhat later in potters and asbestos workers. The distribution of the mottling was found by us to appear invariably first on the right side on a level with the hilus shadow. It certainly becomes quite perceptible on the right side before it appears on the left side and for some time after it does appear on the left side, it is noticeably more marked on the left. In the advanced stage the two sides seem to be about equally involved. The distribution is more or less symmetrical, but naturally not uniform throughout the lung. From the starting point it gradually spreads to the bases and apices, but is never so marked at the extreme apex or base as around the mid-portion of the lung. The appearance of the mottling depends more or less upon the character of the dust. In those exposed to silica the spots are very dense and sharply circumscribed and can be seen when very small. In the case of the less irritating forms of dust the spots are not so sharply defined. The roentgenologic diagnosis in this stage is not difficult. It may be difficult, however, to detect an early tuberculous lesion engrafted upon a well advanced case of pneumoconiosis in the second stage.

The *third stage* is characterized by the appearance of diffuse fibrosis and all that the term implies. While there is no sharp dividing line between the second and third stages, it would appear in some instances that the mottled appearance in the second stage becomes more and more conglomerate and finally passes over into the appearance of dense fibrosis. In other instances a general haze seems to spread over a certain portion of the lung. The greatest density is in the sub-apical regions, although this is not the area of most intense mottling in the second stage. Dense fibrous bands can be seen extending in various directions, and frequently to the diaphragm, causing marked retraction. In this stage the mottling has become extremely coarse and sometimes is to be no longer recognized as such. The heart and blood vessels are frequently dragged out of place and bronchiectatic cavities are quite common.

It is not always easy to differentiate between the fibrosis resulting from tuberculosis and pneumoconiosis and were it not for repeated negative sputum examinations, sometimes extending over a number of years, it would not be easy to say that tuberculosis was not present.

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A CRITICAL REVIEW OF METHODS FOR THE STUDY OF DUST CONTENT OF AIR*

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VARIOUS methods have from time to time been suggested and employed for the study of air dustiness, ranging from mere rough comparative estimations to attempts actually to count the number of floating particles and to weigh the solids in given small samples of air, but until 1916 the number of suggested methods was almost as great as the number of workers interesting themselves in the subject. Early in that year a new method was reported by George T. Palmer (1) of the New York State Commission on Ventilation, and in the next year Dr. E. V. Hill of the Chicago Health Department (2) brought out a new type of dust counter which he demonstrated at a meeting of the American Public Health Association in Washington. Palmer, Coleman and Ward (3) reported a study of three methods with a criticism of fourteen others, comparing results obtained with the Graham-Rogers plate method with the results from the Palmer water spray sampler. The methods referred to but not tested out by them, were Liefmann's oiled plates exposed for definite periods in dusty atmospheres, washed in oil and the washings compared with color standards (6); revolving syrup-covered white paddles (3) read colorimetrically; Aitken's koniscope (7) and his dust counter (8); weighing solids collected from water samples exposed for weeks in the open, as done by the Whipples (9); paper filters washed in oil after exposure to measured air currents and the oil read colorimetrically as done by Rubner (6); cloth filters read colorimetrically after the passage of measured air currents as done by Todd (10); the similar use of collodion wool (Hahn) (6) or resorcin (Winslow and Baskerville) (5) or sugar (Lanza) (11), with subsequent solution of the filtering material and counting of particles; water bubbling as done by Klein for making weight determinations of flue dust (12); weighed cotton wool filters as used by Duckering (13) and Ditman (14); Mariner and Hoskins weighed Soxhlet thimble filters (15) and a modification of Well's centrifugal method of determining bacteria in air as used by the New York Ventilation Commission, with metal bowls substituted for medium-coated tubes (3), but which proved too cumbersome and difficult to operate. They also refer to MacMillan's study of the use of the Cottrell principle (16) of dust precipitation on passage over high tension magnetic poles but give no reference and state no results (3). This last method would be of limited use as the high potential of 40,000 volts would not be readily obtainable, though if a sufficiently compact and portable apparatus were devised containing a transformer, as used experimentally by Cottrell, this method might be adaptable for dust tests. In his early work he used a small transformer to derive a 2000 to 6000 volt current from one of 110 volts and this he found sufficient for experimental work. The apparatus as used by him, however, was not sufficiently portable or compact for general use in dust estimations and there was no way of accurately weighing or otherwise estimating total dust per unit volume of air. Coleman also attempted to use a diffraction device for counting dust suspended in a beam of light and to photograph the luminous particles but obtained no satisfactory results. Dr. Hill reported a series of comparative tests with various methods, found to be more or less unsatisfactory, including cloth screens and vaseline-coated porcelain dishes, as visual comparison or colorimetric tests; muslin filter bags over the inlet of a Sirocco fan, cotton filters connected by a gas meter with a centrifugal fan, and Drechsel wash bottles connected by a meter with an air suction line, as weighing methods: Ait-

*Received for publication May 31, 1919.

ken's koniscope and dust counter, as condensation methods; a diffractiscope devised by himself; and as counting methods, a sugar filter equipped as was his cotton filter, the Drechsel wash bottles, a moistened photographic plate connected with an anemometer and an exhaust ventilator, and his own specially designed apparatus devised as a result of his studies to overcome the defects of the other methods tried. As the result of the former tests the Committee of Standard Methods for the Examination of Air of the American Public Health Association in their final report presented in 1917 (17), recommended the use of the Palmer apparatus as the method of choice for determining dust in air.

As the papers referred to give rather detailed accounts of most of the various methods tested and discussed, it seems unnecessary for the purposes of this article to go into details of these methods again. They may be divided into several groups, as done by Hill, as follows: (1) Visual Comparison; (2) Weighing Methods; (3) Condensation Methods; (4) Diffraction Methods; (5) Counting Methods.

The majority of the methods suggested fall into one or another of these groups and several collection methods may be used to determine dust in more ways than one, as by visual comparison and by weight with a cloth filter, and by weight and count with a water bubbling method. No one method suggested is suitable for use under all conditions and few have any great range of usefulness. Visual comparison methods are the simplest, but at best give only rough estimates of comparative dustiness under similar conditions with similar types of dust, and are very inaccurate when attempts are made to compare dusts of different size range of particles and of different colors or shades of particles. Various methods of air sampling for the purpose of making color comparisons have been used, such as cloth screen, porcelain dishes, oiled plates, paper or cloth filters and syrup-covered saddles, but none of them give even approximations of the actual amount of solids or of the number of particles in suspension in the air, and they have little or no scientific value.

The simple Canton flannel disc filters as used by Todd (10), through which he aspirated 200 cubic feet of air, should prove very useful, however, for rough, easily made comparisons of relative air cleanliness of school rooms or offices, furnishing permanent records very convincing to the layman and effective arguments for improving conditions. The test is similar in principle to the visible dirt test for milk, which has been of such educational value in milk hygiene work.

Diffraction methods based on the luminosity of floating particles in a beam of light, or as used by Hill (2), on the droplets of moisture condensed on such floating particles when air pressure is lowered in a closed chamber, will show all suspended matter, but here also no size differentiation of particles is possible and even ultra-microscopic particles may be included in the test. As Hill states, this method will determine absolute freedom from dust, but comparative tests are hard to make and no absolute quantitative standards can be arrived at. The size of the apparatus as used by Hill prevents the taking of samples large enough to be representative. The same objection holds to the use of the very ingenious koniscope (7) or the dust counter designed by Dr. Aitken (8), both of which employ the same principle of condensation of moisture on floating particles in a saturated atmosphere, when air pressure is lowered. In the koniscope the degree of dustiness is determined by the denseness of the fog as compared with arbitrary standards, a method subject to considerable variation due to the personal equation; in the dust counter the droplets are allowed to fall on a ruled surface and there counted. All suspended particles will be included here and only extreme size variations can be noted. No idea of the sharpness of mineral particles or of their chemical nature can be obtained from either of Dr. Aitken's apparatus.

Weight determinations with properly controlled methods can be made with the greatest degree of accuracy provided sufficiently large samples can be taken, but of themselves give no clear idea of the number, quality or size of dust particles, all of which are very important factors from the hygienic standpoint. Methods employed for determining weight of dusts include the use of cloth filter bags (2), cotton filters (13), sugar filters (11),

(18), wash bottles (2), precipitation in water in open vessels (9), Soxhlet thimble filters (15) and a modification of the Wells centrifugal method (3), and finally the Palmer apparatus (3). A given weight of lead dust per 100 cubic feet will indicate a much less dusty atmosphere than will the same weight of a vegetable or organic dust, and the number of particles in the same weight of similar dusts will depend, of course, on the size distribution of the particles. Whether the weight of suspended matter in the air or the number and the size of the particles is the most important factor depends on the nature of the dust creating substance, its relative solubility, toxicity or density and shape. With soluble or toxic dusts, the total amount of dust likely to be inhaled, and with non-irritating inorganic dusts, the number of particles, is probably the most important factor; while with sharp spiculate mineral dusts the size, distribution and number should both be important.

The sugar filter has been used in two important investigations on dustiness in mines and has given valuable information. Lanza and Higgins (11) in a study of dust in the hard rock mines of the Joplin District in Missouri collected their samples in sugar filters and determined total weight of dust by filtration of the particles in the dissolved sugar. With large air samples the insoluble matter in the clean sugar is usually too small to influence the results materially, but a blank test should be run on a weighed volume of sugar and corrections made if indicated. Lanza and Higgins filtered the dissolved sugar through a Gouch crucible and determined the weight of the retained solids, and observed the nature of the dust particles by mounting some of the dried residue in cedar oil and examining under the microscope. No attempt was made to count particles or determine size distribution. Their method of collection of the samples was by aspiration into their own lungs through the mouthpiece and tubing of an oxygen rescue helmet, with discharge of expired air into a 35 liter Draeger bag. Breathing into the bag was continued until 35 liters of air had been collected. This is the simplest method employed for measuring larger air samples in dust tests and is the only method where the rate of sampling approaches the normal breathing rate of the worker. For this reason, estimates by this method of the amount of dust inhaled by a worker per diem should be most reliable, provided the tests were run long enough to allow for variations in dust production.

Muir and Johnson (18) in their extended investigation of dust in South African Mines also used a sugar filter for collection. Their work is especially interesting as they were the first to attempt an estimation of the relative amount and number of particles of different sizes. These investigators separated their samples from the dissolved sugar into several different groups, classing all particles under 12 microns in diameter as very fine or injurious, and those from 12 to 50 microns as non-injurious. All particles over 50 microns they discarded as too few in number and too large to be of any sanitary significance after sampling. They dissolved the sugar in water to make a 17 per cent. solution and screened this through a 260 mesh wire gauze, the meshes of which average 50 microns in diameter. The filtrate was then separated by an elutriation process, similar to that used in grading sands for filtration purposes (19), into portions containing the two size groups of particles referred to above. This grouping of particles was based on McCrae's work, a part of the same report. He found, on acid oxidation of the lungs of miners dying from pulmonary or bronchial troubles, that the silicious particles recovered were usually less than 1 or 2 microns in diameter and were never over 12 microns. Muir and Johnson made counts by means of the hemocytometer cell of particles from each size group, and also filtered and ignited the residues and weighed the ash to determine weight of mineral matter, ignoring apparently the organic matter present in the dust. They also reported the advisability of substituting a more hygroscopic substance, such as potassium chloride, for the sugar in very dry atmospheres, and in very humid atmospheres a less hygroscopic substance, as ammonium oxalate, cautioning as to the need of more thorough washing of residues before incineration if this was done. For collection of samples they used, as aspirating force, a compressed air suction pump similar in action

to the commonly used water suction filter pump, aspirating 1 cubic foot per minute until 15 to 100 or more cubic feet of air had been sampled.

The excellent work done by Duckering in the English investigations of the lead risk in the tin plate (20), earthen ware and china (13), (21) industries and in woolen manufacture (22) and his careful control of the amounts sampled show the possibilities of the cotton filter. As used by him in his later tests (21), with a carefully calibrated and frequently tested hand pump as suction force, the method is comparatively simple and it is usable anywhere, regardless of the presence or absence of electricity or water power to supply suction. For soluble or toxic dusts, where the important factor to be determined is the total amount of poisonous substance a worker is likely to inhale per diem, it is a simple and very reliable method; but where insoluble dusts, likely to produce more or less of mechanical irritation or trauma and eventually lead to pulmonary fibrosis, are to be estimated, its findings are not so reliable. It retains all particles, including the large ones not likely to reach the lungs, and gives no reliable figures as to size distribution. The shape of the average particles can be studied, however, and some idea of average size determined by microscopic examination, as is shown by Duckering in his tests on woolen dusts (22). In employing this method care must be exercised to dry the filter bottles thoroughly with a current of warm filtered air before and after each test, and to calibrate and control carefully the force of the pump strokes, as was done by Duckering (21).

Dust counting methods have been in use for some time, but here also the amount of air sampled has usually been small, requiring a very large multiplication factor and a consequent large factor of error. As a preliminary to counting, dust may be collected by filtering air through a soluble medium, such as sugar (11), (18), collodion wool (6) or resorcin (5); by precipitating with droplets of condensed moisture, as in the Aitken apparatus (8); by aspirating through a water spray, as in the Palmer apparatus (3); by bubbling through water (2); or by directing a current of air against an adhesive surface, as in the case of the Graham-Rogers glycerin coated plate (4), Hill's moist photographic negative or the varnished coverglass of his newer apparatus (2).

The amount of air sampled in the Aitken counter is measured in cubic millimeters or fractions thereof, and is too small to be representative. For a representative test in a room with little variations in air currents, a number of tests would be necessary in different parts of the room; and in a room with air currents constantly changing in force and direction, tests would need to be taken at frequent intervals to give any approach to accurate results. With the Aitken apparatus this process would be extremely fatiguing as well as time-consuming. This method is extremely accurate as a means of determining the number of particles in a small volume of air, since an actual count is made, based on the particles in 10 cubic millimeters of air, either undiluted for very pure air or diluted from 1:5 to 1:50 for more or less dusty air. This method gives a very much higher count than any other, since it includes the very numerous exceedingly minute particles, too small to be counted by other methods and probably of no significance hygienically. The discrepancy between this and most methods of counting is so great that no comparisons are possible. To determine the number of particles per cubic foot in very pure air, which can be counted undiluted, a multiplication factor of 3,963,100 must be used to estimate the number of particles per cubic foot, as is customary; whereas, with other counting methods, which make no attempt to count particles at the limit of vision, only decidedly dusty atmospheres give counts ranging in the millions. Ordinarily samples taken with the Aitken apparatus must be diluted from five to fifty times with filtered air for counting, this requiring additional multiplication of from five to fifty, bringing the totals up to hundreds of millions, more than is ever found by other methods. In addition to this factor of error, the small size of the sample and the fact that it is taken at a given instant, rather than during a time interval sufficiently long to allow for normal variations in dustiness due to varying currents and varying dust production in industrial processes, vitiates the value of a single test. This necessitates the wearisome tak-

ing of many tests in different locations and at different times, if one is to draw any conclusions from the results of tests. The Aitken counter indicates only the total number of particles present, giving no indication of relative size, shape, weight or chemical nature. Dr. Aitken's dust counter, as used by him, in studying the effects of air currents and of pollution by combustion products on dust distribution in the meeting room of the Royal Society of Edinburgh and in studying the effects of temperature and humidity variations on air dustiness in the open at Colmonell, give valuable information more easily obtained than by any of the other methods of air sampling suggested. The particular value of the Aitken dust counter is in the making of the count at the time of collection and directly in the apparatus in which the air sample is obtained.

With the Hill apparatus a decidedly larger sample is taken, air being drawn by a hand suction pump against a specially prepared varnished cover-glass. The volume of the sample ranges, according to Hill's directions, from 20 to 80 cubic inches, based on the capacity of the pump chamber and the number of strokes, but is reduced to from 10.0192 to 25.048 cubic inches when corrections are made for pump leakage, area of cover-glass counted and estimated efficiency of the method. The particles adhering to the cover-glass are counted under a low power microscope lens and here relative size and shape or particles can be seen, though Hill in his original article and on his "synthetic air charts" makes no reference to size differentiation. For the purpose of testing air washers delivering a steady current of air, this method should give very satisfactory results very simply and easily obtained, provided the percentage efficiency of 62 per cent as stated by Hill is correct, but this should be carefully tested and frequently checked. Whether the cover-glass retains an equal percentage of total particles in all instances is very doubtful. Where, on an average, only 62 per cent. of floating particles are retained in the first capsule, this percentage would certainly vary greatly, depending on the degree of dustiness, the force of the pump stroke and the specific gravity and size distribution of the dust particles. In calculating the volume of air aspirated based on the pump capacity, Hill makes an allowance of one-tenth volume for "leakage and space occupied by plunger rod" but the amount of leakage would depend somewhat on the force of the stroke and this is not controlable. He also counts particles not on the entire three-eighths of an inch cover-glass but on the portion "within the limits of the square of the ocular," which he figures as 80 per cent. of the cover-glass when using a No. 3 objective and a No. 1 eye-piece. The unequal distribution of particles, as shown in his photographic illustration, introduces another possible source of error, and the taking of only one sample at a time prevents the making of an average of a number of counts to correct this error as can be done with the Palmer method. For testing dwelling room, office or school room air with no continuing marked source of added dust, fairly accurate relative counts might be made if care were taken to use approximately the same speed of piston stroke in each case and without this if all six capsules supplied were used in tandem (but this would increase materially the work of the test), provided tests were taken in various locations in each room and an average or range determined. For the purpose of testing dust conditions in dusty trades and formulating standards for the same, this method also takes too small samples and samples during too short intervals to allow for changing conditions of dust production and variations in air currents. One obvious advantage of this method, where it is otherwise suitable, is the portability of the apparatus, its simplicity and independence of electric or other mechanical motive power for suction. Also the direct obtaining of a permanent record by mounting the cover-glass is of distinct value.

Dr. Graham-Rogers' method of aspiration of a measured volume of air past a glycerin covered plate is somewhat similar in principle but requires a motor of some kind, preferably electric, to supply aspirating force and a measurable air supply. On the other hand, larger samples can be taken and the sampling time is extended into periods of minutes. Graham-Rogers, himself, states that this method allows a greater proportion of smaller sized particles to escape and entrains rela-

tively more of the larger sizes. Here also the total number retained as well as the proportion of the different sizes depends on the force of the suction as well as on the character of the dust. Palmer, Coleman and Ward have shown that this method is distinctly less accurate than the Palmer apparatus and gives consistently lower counts.

As pointed out by Palmer, the use of sugar or other soluble filtering substance for entraining dust for counting is limited by the slowness of aspiration necessary, due to the resistance offered by the filtering material, and the possibility of there being insoluble particles already present in the sugar, though the error due to this latter factor is a small one in dusty atmospheres. The presence of the filtering substance in the solution may interfere with any chemical tests to be made on the dust or on the differentiation between organic and inorganic residue, as can be done with samples obtained with the Palmer apparatus, though thorough washing of the residue after filtration may remove this difficulty.

Where water bubbling is used (2) samples of several liters in size may be taken, but unless the bubbles are small and the rate of passage slow, many of the smaller dust particles will pass through in the bubbles and will not be included in the test. This method necessitates a suction pump of some type with a meter to measure air flow. Aspirating bottles give too slow a flow to enable a large sample to be taken, and a suction fan with direct connection, as in the Palmer method, would give too rapid a flow to entrain much dust.

The Palmer apparatus makes use of a small electrically driven centrifugal fan to aspirate air through a sample of water in a specially designed glass bulb, so constructed that the air is drawn through a fountain spray curtain of water which retains by actual test 98 per cent or more by weight of finely divided dust added at the intake. A rheostat controls the strength of the pull and the rate of flow is measured by a gasolene manometer connected with a venturi throat at the inlet as in the first models [see illustration in Palmer's article (3)], or between the water and the fan, as in the later models. The first apparatus described in the American Journal of Public Health had several mechanical defects which have been remedied in the later model, as supplied now by Wallace and Tiernan, but the principle has not been changed. The new apparatus is more compact and more rigid in its wooden case than was the first in a leather dress suit case. The outlet from the fan is placed on the top and not on the side near the intake, as at first. The latter position is much less likely to interfere with the air supply to the intake. The rate control by means of the sliding valve over the outlet to increase resistance is simpler and more easily regulated than the rheostat control in the first model, and, lastly, the collecting bulb is more easily detached for emptying and washing and the manometer is subject to less functuation than formerly. The efficiency of the apparatus as supplied for use with a 110-volt, direct or alternating current can be much increased for industrial work by the introduction of a four-point switch connecting with a 110-volt lamp in series with the motor, since many industrial plants making their own current supply a 200 to 220-volt current, which cannot be used with the apparatus as supplied by the manufacturer.

In using the Palmer apparatus in industrial tests, it is set up with the intake as nearly as possible at the level of the worker's head and so the sample will represent the normal dustiness to which the worker is exposed. After making the proper electrical connections, the motor is started and the fan run to aspirate five cubic feet of air per minute, the optimum rate for efficiency of the apparatus, and so run for from five to forty minutes, depending on the degree of dustiness and the turbidity observed in the water in the collecting bulb. From time to time fresh water must be added to replace loss by evaporation but this can be done without interrupting the test. The rate of evaporation is, of course, dependent on the relative humidity of the room and the need for more water can be told easily by an increase in rate as shown by the manometer scale. At the end of the collecting period the motor is stopped, the bulb disconnected and its contents discharged into a sampling bottle, best calibrated at the 100 c.c. mark, and the bulb rinsed out several times with fresh water, which is then added to the sample. To avoid any necessity for correction of results based on a control test of the water,

it is best to use distilled water filtered through a Berkefeld filter so as to be practically free from suspended matter and free from ponderable residue on evaporation. The residue from 100 c.c. of distilled water is negligible for the purposes of the test. For making the count, 1 c.c. is taken and either placed directly in a Sedgewick-Rafter cell or diluted, according to the turbidity of the sample. Dust counts are made with a ruled eyepiece and a low-power lens, counting the particles in ten areas, each representing the dust in 0.25 c.mm. of water. With 100 c.c. samples the total of the ten counts can be multiplied by 4000 and divided by the number of cubic feet of air sampled. If this is 100 cubic feet the multiplication factor thus becomes 400.

Owing to the volume of the air sampled and to the fact that the duration of the sampling period allows for normal variations, a fair estimate can be made of the amount of dust likely to be inhaled by a worker in his day's work. An average sample takes from 50 to 100 or 200 cubic feet of air during from ten to forty minutes. With a tidal air of 30 cubic inches and from 17 to 18 respirations per minute a man would inhale about 18 cubic feet per hour, or 144 cubic feet in an eight-hour day. With these figures as a basis, the total dust inhaled per diem and also the amount of poisonous dust inhaled can be easily estimated, as was done by Miller and Smyth (23). No comparative figures are available showing the relative efficiency of the Hill and the Palmer methods, but Hill's averages of 500 to 1500 particles per cubic foot for clean outdoor air and 10,000 to 30,000 per cubic foot in a mechanically ventilated schoolroom without air washers are far below any figures recorded for the Palmer apparatus, and are nearly in the same range as Palmer's figures obtained with the Graham-Rogers plate.

The Palmer apparatus owes its great superiority to the other methods of dust sampling so far devised, to several factors:—First, the size of the sample obtainable and the fact that collection can be continuous over sufficiently long intervals to allow for normal variations of dust producing and dust distributing conditions; second, the fact that sufficient dust is collected and collected in such a form that various methods of quantitative and qualitative examinations can be carried out on the same samples. This latter fact is a great advantage especially in the study of industrial dusts. No one method of examination can give us all the information necessary to correctly estimate the injuriousness or inocuousness of existing conditions of dustiness in any industry. With the Palmer apparatus counts can be made of the total number of particles in a given volume of air and the relative proportion of large and small particles can be determined. The nature of the particles can be noted, whether fibrous, amorphous, spiculate or crystalline. Solubility can be tested by comparative tests with water and with an oily or other collecting medium, as is being done at present in an investigation directed by the author. In similar processes, fair comparisons of dustiness can be easily made from turbidity readings of samples representing equal amounts of air (23), though this cannot be done with any degree of accuracy with dusts of different chemical or physical nature. The weight of dust in the sample can be determined by filtering 50 to 100 c.c. of the water through a Gouch crucible and drying to a constant weight, as recommended in the Standard methods (4), though this allows to escape unrecorded any water-soluble matter present in the dust, such as lead salts in lead industries, or any soluble organic substances which may be present, as in tests, not yet published, carried out under the author's direction in a plant manufacturing synthetic indigo. To insure the inclusion of soluble dusts in the total weight, a given amount of the water sample may be evaporated to dryness and the residue weighed, and it may then be incinerated to separate organic from mineral dusts. Sufficient sample can be obtained to permit of chemical tests for toxic or other substances present in the dust which might affect the health of workers, as was done in several instances by Miller and Smyth (23) (24). Permanent samples can be preserved or photographic records can be made showing the nature of the particles. By comparing count with weight and size distribution, an idea may be had as to the relative specific gravities of different dusts.

The Palmer apparatus is portable, easily set up, can be run at a fairly constant

rate and single tests give much more information than do single tests or multiple tests with any other method. With the addition of a 110 volt light bulb, as mentioned above, it can be run on any lighting current likely to be met with in industry, but an electric current is an essential and the apparatus is useless where there is none. Accurate counting requires some practice, and if size differentiation of particles is attempted different observers will vary somewhat in their counts, though after a little practice no great variation was noted by Miller and Smyth in their work (23). Size differentiation may be inaccurate with markedly hygroscopic dusts and some of these may show a tendency to cake and make the breaking up of clumps by shaking difficult or impossible. Miller and Smyth found this trouble in sampling cement dust which on standing in the water hydrated to form cakes or flocculent masses. Hill speaks of the likelihood of violent shaking of samples in liquids breaking up clumps to give more particles than were present in the air, but the author doubts if this causes as much error as would the clustering of particles on an adhesive surface such as Hill uses for collection. Dust counts can include all particles, or may exclude long fibres or large particles not likely to reach the bronchi and include only particles averaging one-fourth standard unit (.0001 sq. mm.) in area, as those most likely to be of interest hygienically.

Of the various methods proposed from time to time for the study of air dustiness, few have any great range of usefulness and a number are extremely inaccurate as absolutely quantitative tests, though all have some value as comparative methods when used under similar conditions with dusts of like nature. Colorimetric methods particularly are useless for comparing unlike dusts, as coal or soot and clay or textile dusts.

Condensation and diffraction methods are extremely accurate for minute quantities of air and experimentally to determine absolute freedom from floating matter, but are impractical for industrial dusts and conditions associated with changing air currents and varying dust production.

For estimating the total amount of dust, as, for example, the amount of coal and soot, precipitated over a given area, the simple method of collection in open vessels containing water as used by the Whipples gives information obtainable in no other way, but it gives no idea of the amount of dust suspended in the atmosphere at a given time. Weighing methods alone do not give complete information and do not indicate by themselves the nature of the dust or the size or number of the particles. Their special field of usefulness is in testing soluble and poisonous dusts.

Counting methods as used vary greatly in accuracy and in total numbers of particles counted by different methods. The Aitken counter gives the highest results but gives little size differentiation and includes ultramicroscopic particles of no hygienic significance.

Hill's and Graham-Rogers' methods give very low counts and apparently exclude most of the minute but visible particles which on account of their size and weight are most apt to reach the bronchioles and air cells and produce pulmonary fibrosis in the worker. For testing air washers and possibly for schoolrooms and offices, Hill's apparatus gives valuable information and certainly is the simplest and easiest method to operate, and requires no electric current or outside suction force.

High tension magnetic field dust-precipitation is not yet a practical method, owing to the lack of conveniently portable sampling apparatus supplying the high tension current essential to the process.

The sugar filter as used by Muir and Johnson (18) gives more information than does any other method except the Palmer, but requires more care and skill in carrying out, and there is more chance for error in the processes of solution, filtering, elutriation and counting. In one detail their method makes a determination not made by the Palmer method as recommended—*i.e.*, in the determination of the weight of the smaller particles up to 12 microns, though this determination could readily be made on the water sample from the Palmer sampler using the same technic as that employed by Muir and Johnson.

For range of usefulness and adaptability to varying conditions and for the amount and variety of information furnished, the Palmer water spray sampler with the modification proposed and used by the author gives results unobtainable

by any other method so far suggested. A single sampling will furnish data for determining the amount of dust by weight in a given air volume, the number of dust particles per unit volume, the size distribution of particles, the relative volume of dust as shown by turbidity readings on water samples, the relative amounts of organic and inorganic matters in the dust and the physical and chemical nature of the particles.

With the aid of tests made with this apparatus, the U. S. Public Health Service, Division of Industrial Hygiene and Medicine, are endeavoring to formulate maximum standards of permissible dustiness in various industries, based on the count and weight of samples and the physical and chemical nature of the dusts encountered. Tentative standards for the abrasive industry were suggested by Professor C. E. A. Winslow in a paper read by him before the Industrial Hygiene Section of the American Public Health Association held in Chicago in December, 1918 (25).

Conclusions

1. No single method of air sampling for dust content, as yet devised, is ideal but several methods are particularly adapted to special conditions, and a number have distinct fields of usefulness.

2. The essentials of a satisfactory method for making a quantitative dust test are: (a) an aspirating force capable of accurate measurement of the amount of air aspirated per unit of time or per pump stroke; (b) a receptacle for retaining dust particles which will retain practically all of the particles from one-half to 50 microns in diameter; (c) a satisfactory method of making the required tests on the sample as collected.

3. In studying industrial dusts sampling must cover sufficient time intervals to allow for normal variations in dust production and dust distribution and collections should be made as nearly as possible from the working level. The larger the sample and the longer the period of sampling the less the probable errors of the method.

4. With dusts containing poisonous substances, samples must be large enough to permit of accurate chemical tests.

5. For studying industrial dust conditions weight determinations alone do not give sufficient information, except possibly with soluble poisonous dusts.

6. For complete studies and for fixing of permissible limits of dustiness, tests must permit of estimating weights and counts and determinations of the physical and chemical nature and size of dust particles. These tests are at present best and most easily made on samples collected with the Palmer apparatus, though the sugar filter samples treated according to Muir and Johnson's technic give as much information.

7. Other tests of distinct value are colorimetric tests with cloth screens, the Hill capsules and pump, the Aitken dust counter, the cotton filter as used by Duckering, and the precipitation method of the Whipples.

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BACK STRAIN—AN ACCIDENT OR A DISEASE?*

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THE specialty I represent appreciates deeply, gentlemen, the opportunity of participating in your scientific sessions. Orthopedic surgeons serving with the A. E. F. were trusted with the working out of certain problems. These problems, roughly speaking, were the lesions of the extremities and spinal column, both pre-combat and post-combat. Greatly helped by this experience, the specialty is eager to be of more service in peace, convinced that there is no essential difference between certain static and traumatic conditions of civil life and the lesions of the extremities and spinal column which result from pre-combat and combat army life. Perhaps the majority of these lesions in civil life are the results of industrial conditions and industrial accidents. No symptoms are more common, none are less intelligently treated, than those grouped under the term of "Back Strain." You have given me a subject in the form of a question. I do not believe the question can be answered simply, but in the time allotted an attempt will be made to indicate the lines along which the analysis of any given case must proceed and to demonstrate some of the factors which contribute to the complexity of the solution. We shall discuss, briefly, the so-called static conditions which are known to give rise to the symptoms of back strain. Then by means of lantern slides we shall show you a few of the common and more typical lesions and try to emphasize the fact that combinations of structural abnormalities with static or traumatic strains are frequently found. We shall suggest that the complete relief of these symptoms is often accomplished only by appreciating that the recent and perhaps trivial strain may be superimposed upon a structural defect, which up to the time of the accident had been unsuspected and unfelt.

I. STATIC CONDITIONS

Under this heading we shall group the symptoms that may occur as the result of:

(a) Faulty posture acquired probably in childhood and accentuated in adult life.

(b) Faulty positions in which work is done.

(c) The combination of some pelvic disease, vertebral abnormality or acute strain with faulty posture or a faulty position of work.

(a) Faulty Posture.

These postures mean faulty mechanics for the erect position and a lessened resistance which results in fatigue amounting at times to acute strain. We must not forget also, that foot-strain often gives rise to back pain as the back muscles attempt to accommodate.

(b) Faulty Positions of Work.

The tiring effort of the neck and lower back muscles to assist a poor occular focus is familiar to the ophthalmologist; the stenographer's chair which offers no support to the back; the low desk of the draftsman or the low bench of the workman; the cramped positions of miners in the low coal galleries may serve as examples of occupational back strain and need no comment.

(c) The obscure occupational backs are those which fail to completely recover when postural strain is relieved by supports, by the correction of vision, or by correcting conditions of work. Rest surely relieves, but symptoms recur and we must look for combinations or for complications of pelvic or prostatic disease, arthritis or congenital abnormalities.

It has long been recognized that sciatica is less definite a diagnosis than rheumatism, and in nearly all low-back lesions, it is a common symptom and usually only a symptom. It may be associated with tenderness along the course of the nerve or portions of the nerve. Almost never in a so-called sciatica do we find the muscular weakness which accompanies a true neuritis. In our opinion, a true localized neuritis of the sciatic nerve is as rare as that of the other main trunks.

*Read before the Fourth Annual Convention of the American Association of Industrial Physicians and Surgeons, Atlantic City, June 9, 1919. Received for publication June 14, 1919.

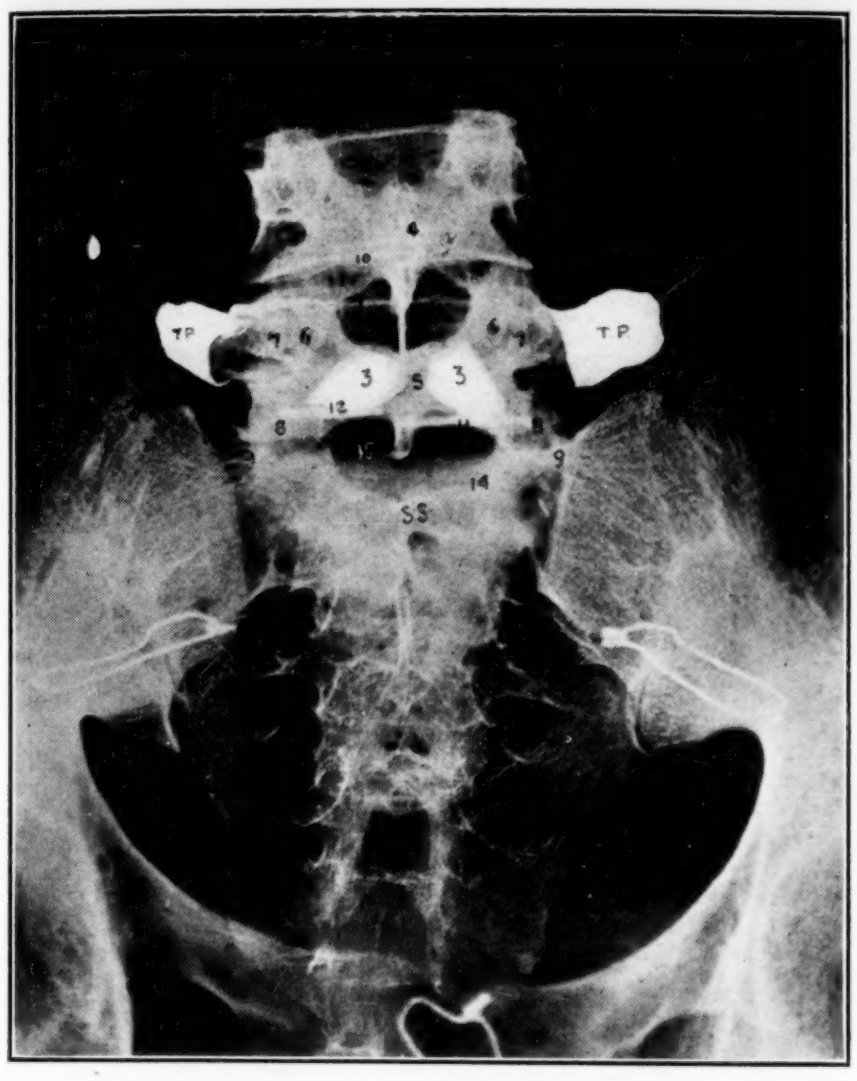

FIG. 1-a.—Reconstructed lumbo-sacral and sacro-iliac region in accordance with supposedly normal standards. (See key.)
Kindness of Dr. Arial W. George, Boston.

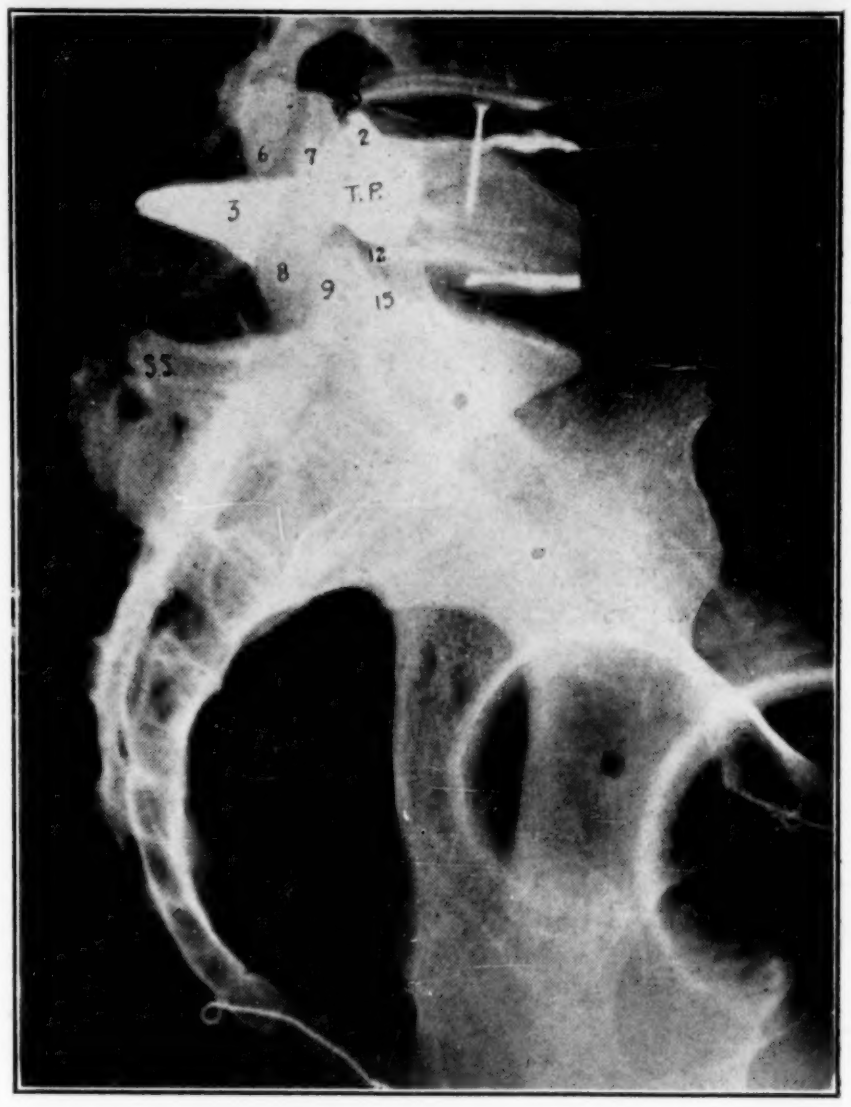

FIG. 1-b.—Reconstructed lumbo-sacral and sacro-iliac region in accordance with supposedly normal standards. (See key.)
Kindness of Dr. Arial W. George, Boston.

KEY

1. Anterior superior border, 5th lumbar vertebra.
2. Posterior superior border, 5th lumbar vertebra.
3. Lamina, 5th lumbar vertebra.
4. Spinous process, 4th lumbar vertebra.
5. Spinous process, 5th lumbar vertebra.
6. Inferior articular process, 4th lumbar vertebra.
7. Superior articular process, 5th lumbar vertebra.
8. Inferior articular process, 5th lumbar vertebra.
9. Superior articular process, sacrum.
10. Anterior border, 3rd lumbar vertebra.
11. Anterior inferior border, 5th lumbar vertebra.
12. Posterior inferior border, 5th lumbar vertebra.
13. Posterior inferior border, 3rd lumbar vertebra.
14. Prominence of the sacrum.
15. Superior border of the sacrum posteriorly.
ss. Spinous process of sacrum.
tr. Transverse process.

These numbers correspond in the antero-posterior and lateral views.

II. CONGENITAL ABNORMALITIES
(Figs. 1, 2, 3, 4 and 5)

Until Böhm and Goldthwait focused attention on the extreme frequency of the congenital abnormalities of the low spine, we had been recognizing only the gross lesions of wedge shaped vertebrae or a marked spina bifida or an occasional cervical rib. Since we have been scrutinizing the X-rays of lumbar spines and sacro-iliac joints we find it rather difficult to determine a standard for the normal in this region, so varied in shape are the elements. Reviewing hundreds of plates taken in the Massachusetts General Hospital, for all sorts of conditions including suspected kidney, bladder, pelvic and prostatic lesions, less than half could be said to show completely normal bony outlines, even using the term with generous latitude.

Many of us here present would show these abnormalities and it is to be hoped we will remain in ignorance of their existence. Some of them are sources of strength rather than weakness, but once an accident occurs which produces a strain, we must be sure that their existence does not complicate the lesion or call for a mechanical treatment which must take cognizance of them and may differ from the mechanical treatment indicated in a so-called normal case.

III. ACCIDENTS

(a) Simple muscular or ligamentous strain.

Muscular strain is probably the com-

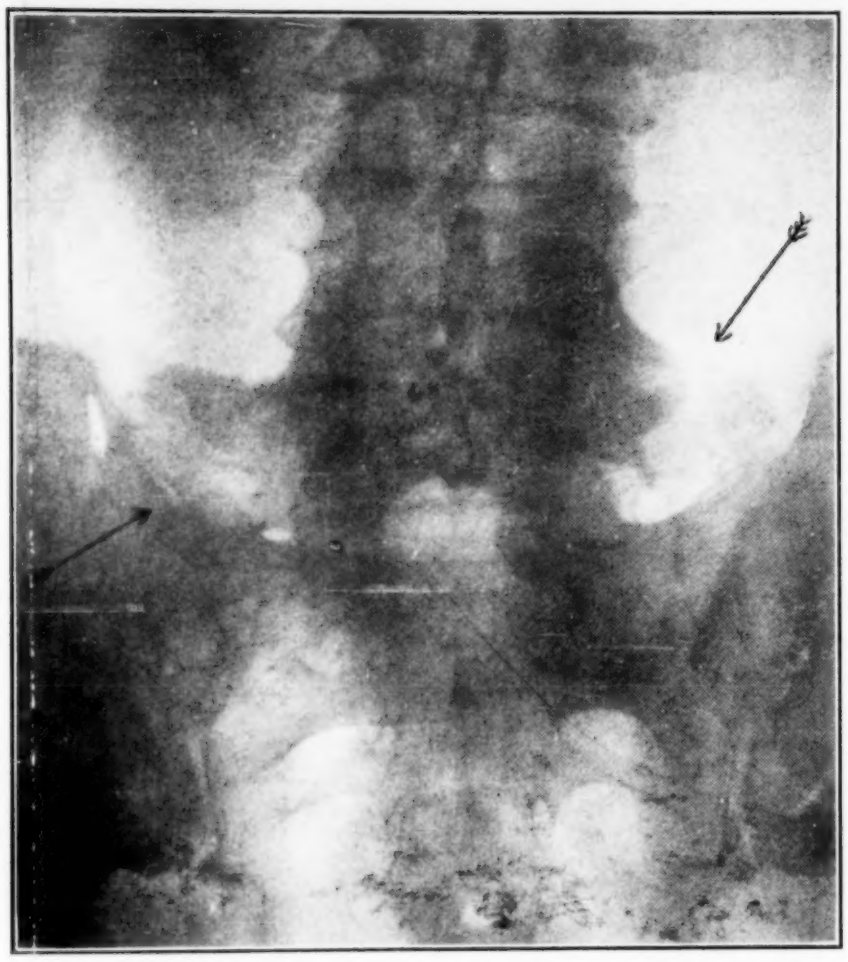

FIG. 2.—Abnormally shaped transverse process of the last lumbar vertebra suggesting an articulation between it and the top of the sacrum. Hypertrophic changes between the 4th and 5th lumbar vertebræ.

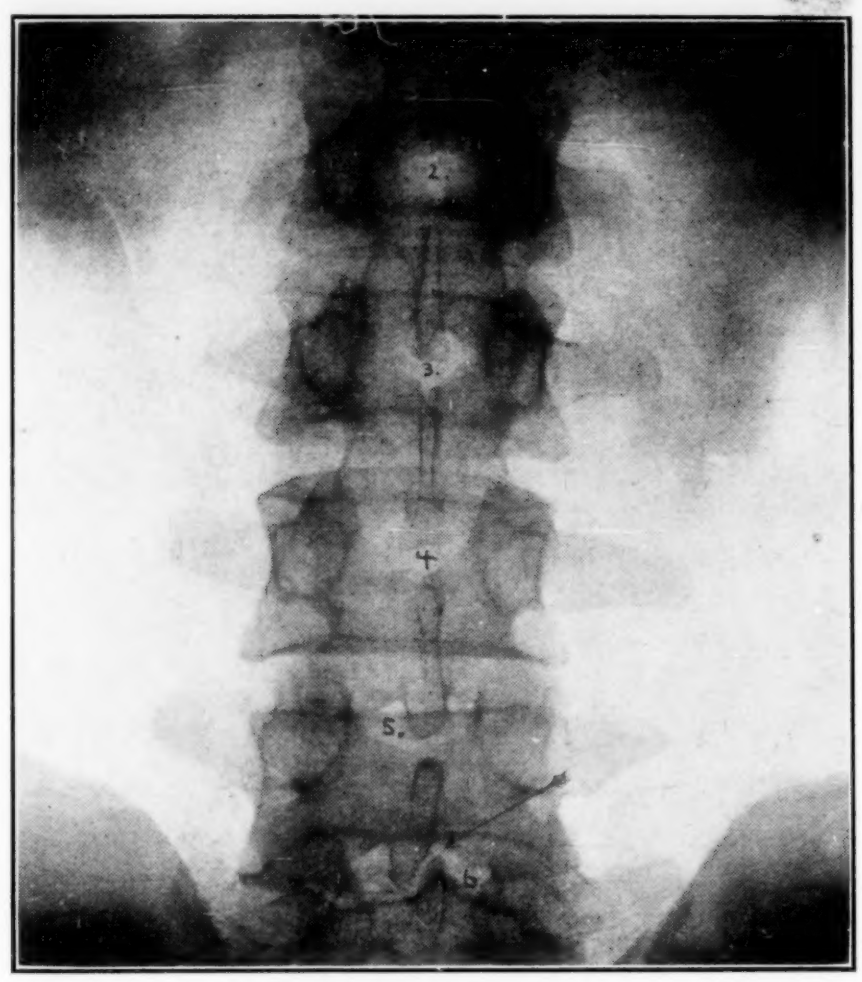

FIG. 3.—Six lumbar vertebræ. Note articulation between spinous processes of 4th and 5th lumbar.

monest industrial lesion. Recovery is usually quick under simple rest, whether or not this rest is accompanied by the use of the well-advertised liniments to which the recovery is usually ascribed. Sometimes the symptoms from these traumatic lesions are persistent. A direct blow may seriously damage muscle fibres or a hematoma may leave unusually strong adhesions after it has been organized. A rupture of a few fibres of an origin or insertion to a spinous or transverse process, or of the attachment of the lumbar aponeurosis to the sacrum or iliac crest, may continue to be a painful or irritating spot, analagous to the discouraging tennis elbows and golfers' wrists of civil life. Complete immobilization for six weeks, followed by a gradual but persistent use, is often the shortest road to recovery in back lesions of this sort, as it is in tennis elbows.

(b) A true sprain of the strong vertebral interspinous or articular ligaments or a tear of a portion of the lumbar aponeurosis is more rare and more serious. These lesions are sharply localized and require efficient immobilization. The lumbo-sacral and sacro-iliac ligaments seem to be subject to strain, to which they yield more commonly than the others. Cases are on record of the rupture of the intervertebral disc with the protrusion of the corpus spongiosum causing an actual pressure on the cauda equina. This must be extremely rare.

Sciatica is again a very frequent symptom of these lesions, especially of the lumbo-sacral and sacro-iliac group.

(c) Ligamentous rupture or relaxation associated with displacement.

FIG. 4.—Partially sacralized last lumbar vertebra, causing extreme asymmetry of the sacro-iliac joints and the articulation between the 4th and 5th lumbar.

Slight displacements.—We must admit, I believe, the existence of vertebral displacements or mal-alignments, sometimes demonstrable by the X-rays and sometimes not. I am personally in doubt whether these displacements are connected with the symptoms as cause or as effect. The frequent marked and symptomless mal-alignments of scoliosis suggest that they are usually the effect. Nothing can persuade our friends of the osteopathic school, that the true cause is not the slight displacements which they honestly believe they can palpate and correct by repeated manipulations. The fact that the symptoms frequently disappear under their treatment is proof enough to them. However, after an entirely honest and fairly thorough attempt, sympathetically aided by some of their best practitioners, I have been unable to satisfy myself of this causal relation, nor have I been able to prove conclusively the existence of the displacements which they were certain they could palpate.

Spondylolisthesis. (Figs. 6 and 7.)—The relaxed lumbar spine, lumbo-sacral or sacro-iliac joints, pivotal as these structures are in the erect biped, may quite as well give symptoms as relaxed knee joints or relaxed feet. It is a question of the strength of the supporting muscles. The contortionist as a rule is free from back strain. If one or two structures in a joint mechanism are weak while the others are strong, we shall surely have symptoms. The typical example of this is in the lumbo-sacral joint, with the thrust of the body weight ever tending to push the fifth lumbar forward in front of the sacrum. We do well, however, to remind ourselves of Sir Arbuthnot Lane's comment on the scholarly monograph of Neugebauer on spondylolisthesis, reporting a comparatively small number of supposedly unique specimens. Lane said "Every coal heaver I ever dissected had spondylolisthesis." The lesion is not difficult to detect. In backs with poor musculature, it is a cause of constant or recurring discomfort, and it is not easy to relieve speedily without a fixative operation on the spine.

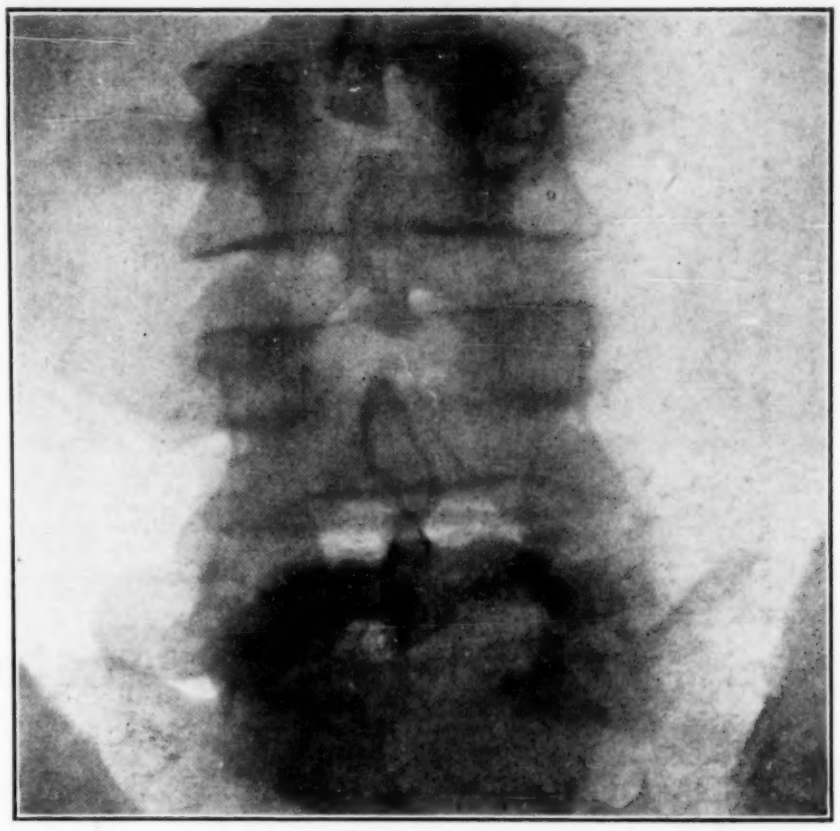

FIG. 6.—Abnormal position of the 5th lumbar suggesting an early spondylolisthesis. Note the impingement of the spinous processes of the 4th and 5th lumbar vertebræ.

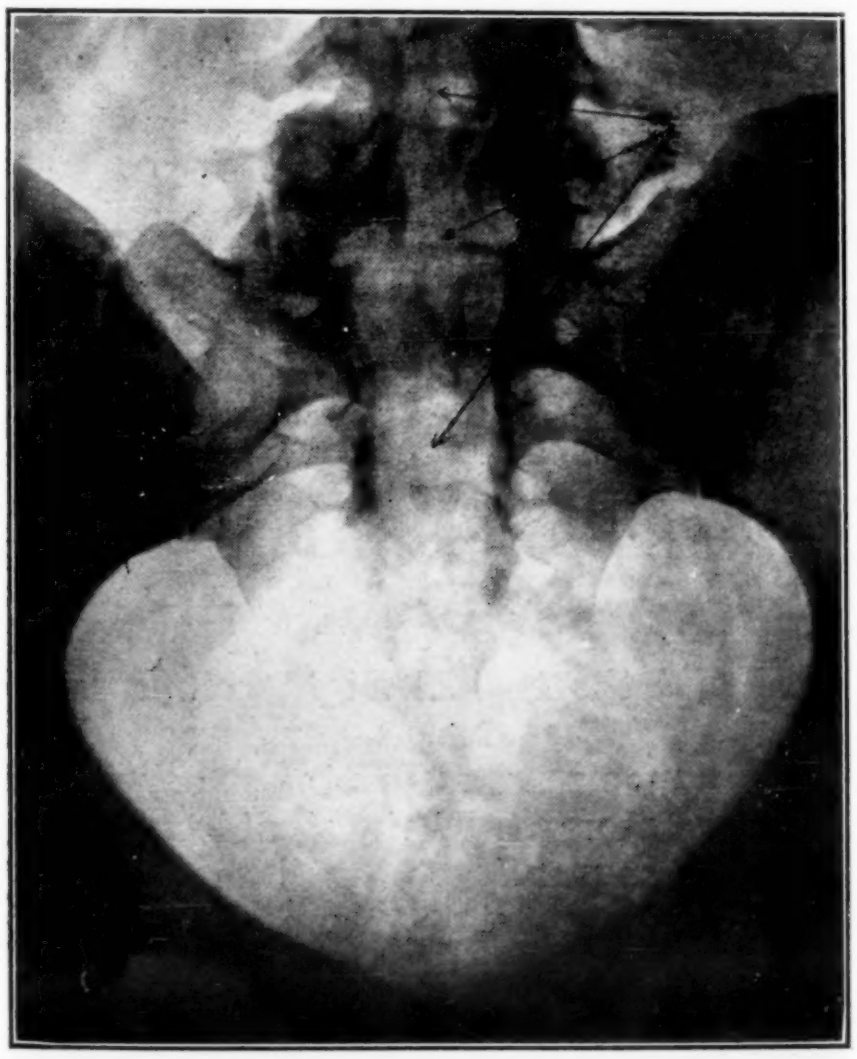

FIG. 5.—Spina bifida. Failure of neural arch to close in last two lumbar vertebræ and entire sacrum.

Sacro-iliac lesions.—Although I admit without regret that I was a partner in what I believe was a sort of a rediscovery of displacements and relaxations of the sacro-iliac joint as a common cause of back strain, no one regrets more than Colonel Goldthwait and I the carelessness with which the diagnosis of "sacro-iliac disease" is made today. It does, of course, exist—tuberculosis, chronic arthritis, malignant disease—but it is no entity and as the term is being used, it

FIG. 7. — Complete spondylolisthesis. Note shadow of body of 5th lumbar vertebra completely anterior to sacrum, its superior surface facing forward.

means nothing. That the normal joint has motion in it, can be demonstrated by dissection or by stereoscopic X-rays. That the amount of motion is variable in different individuals is a fact also. That lack of normal motion is often quite as likely to give symptoms as hypermobility is probably true. Actual displacements, slight and considerable, do occur. Many of them are reducible in their early stages by forcible manipulations at one sitting with or without an anaesthetic, or by repeated less strenuous active or passive movements. Old, irreducible displacements often become symptomless. If a sacro-iliac joint is to be protected, an accurate knowledge of its anatomy is necessary. Lateral pressure on the iliac crests springs it apart. The Italian laborer, when he digs, shifts his belt from his waist line to the level included between his trochanters and his anterior superior spines. He has learned his anatomy, if not the nomenclature, from his symptoms.

Sciatic scoliosis is a term almost as nondescriptive of the cause as sacro-iliac disease. Various lesions of the muscles, of the lumbo-sacral and sacro-iliac joints may give rise to the phenomenon. Most of the literature under this heading is German and the best discussion of the symptom-complex and its varied treatment in English, is by Bucholz, writing from the clinic of the Massachusetts General Hospital.

Sciatic pain again in all these lesions is a frequent precursor of the diagnosis, a concomitant of the recognized lesion or a persistent sequela of the reduced displacement.

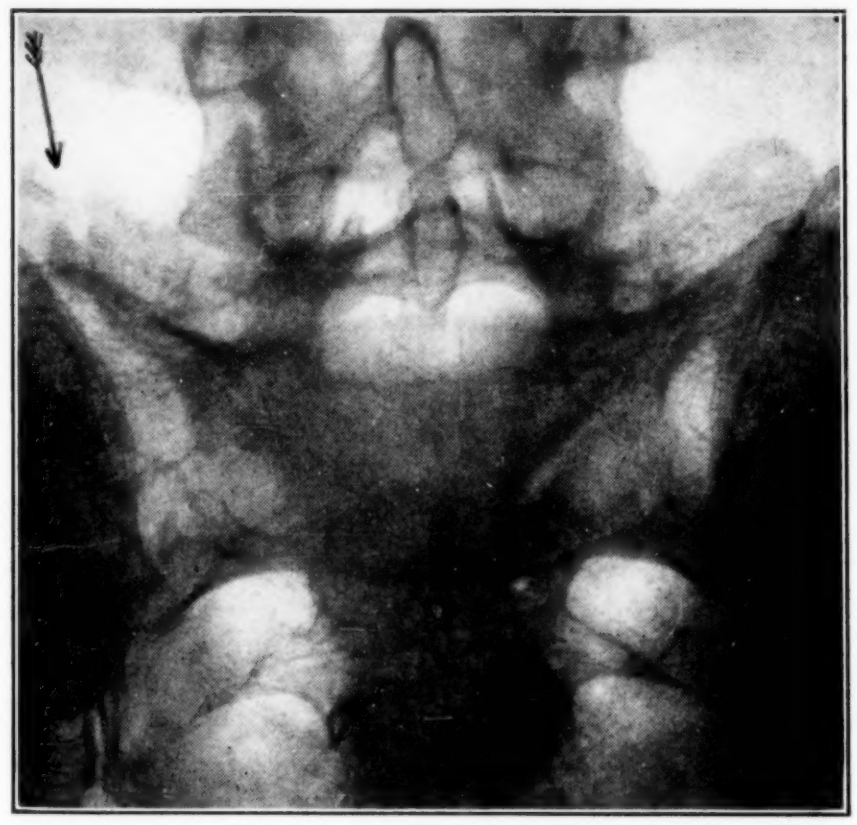

FIG. 8.—Fracture of tip of transverse process of the 5th lumbar vertebra.

(d) Fractures. (Figs. 8 and 9.)

(1) Fractures of the vertebral processes and articular facets, or of the transverse or spinous processes are not as uncommon as is their diagnosis without the X-ray. Fracture of an articular process or processes frequently leaves a region of rigidity and may give pressure symptoms from the accompanying effusion. Fractures of the transverse or spinous processes recover if immobilized, but often remain un-united and painful if not recognized and not treated.

(2) Fractures of the bodies are also much more common than we have believed and the typical slight compression fracture of the spongy vertebrae which firmly impacts is surprisingly devoid of early symptoms. Their significance lies in the subsequently stiffened area and in the tendency to slow over-production of bone which frequently takes place as the result of unprotected activity. This so-called Verneuil's disease may produce an irritative myelitis whose onset may be months or even years after the original

trauma. By early and rather prolonged immobilization the danger is often escaped. Operative immobilization of the area may well be the safest treatment in the laboring man.

(3) A word should be said here again concerning the importance of recognizing the existence of a congenital abnormality or an acquired disease in connection with the traumatic lesion. Different leverages are frequently produced and adjustments of the weight-bearing mechanism and muscular action are necessary. This new condition, plus the old lesion, requires nice appreciation of the mechanics, if relief from weakness and pain is to be obtained.

IV. DISEASE

(a) Lumbago. We will leave the acute myositis, which perhaps alone ought to be allowed this name, to the internists. The name is perhaps not as frequently misused as sciatica, but its abuse is still a reproach.

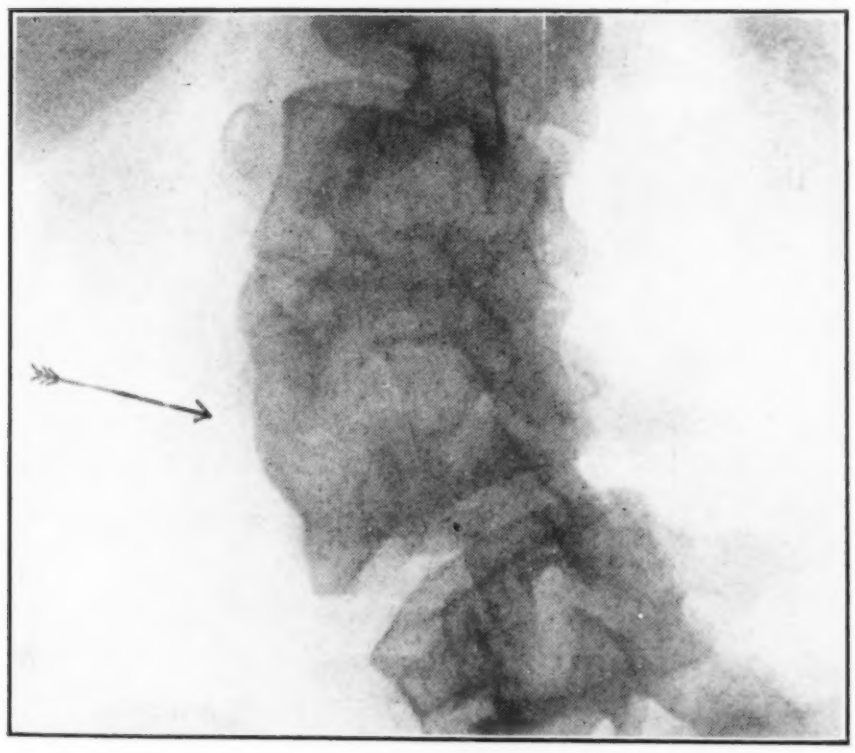

FIG. 9.—Asymmetrical fusion of the 3rd and 4th lumbar vertebræ; probably congenital abnormality or old compression fracture.

(b) Non-tubercular arthritis of the spine is of various types. (Fig. 10.)

Acute infectious arthritis sometimes accompanies a Neisser infection or a typhoid. We see, not infrequently, the chronic poker backs of the so-called Strümpel-Marie or rhizomyelitic type, in which the articular processes first anchylose and the spine and root joints of the shoulder and hip become fixed by bony union.

More common by far in persons beyond middle age, is the hypertrophic type with beak-like processes starting from the edges of the bodies of the vertebrae and involving frequently the articular processes and sacro-iliac joints. It frequently gives rise to few symptoms other than diminished flexibility, though it rarely produces complete anchylosis.

(c) Tuberculosis (Figs. 11 and 12) in the form of Pott's disease is far less easily diagnosed in the adult than in the child. Our own failures to make the diagnosis have, we trust, led us to be very wary. There is often no kyphos and there are often few typical symptoms. The lesion and the surrounding abscess shadow are discovered at times only by the X-ray. When a disease attacks the sacro-iliac joints, it is often most misleading and with our present greater familiarity with non-tuberculous lesions of these joints, we are often likely to forget the very real possibility of these more serious conditions.

Under appropriate rest by recumbency and mechanical or operative measures, tuberculosis of the sacro-iliac joints is by no means always fatal, and recovery is frequently extremely satisfactory. The seemingly healed tuberculous lesion is not unlikely to have exacerbations for many years following any slight trauma. Painter's paper on the malignancy of bone tuberculosis has a distinct bearing on the prognosis, especially in its relation to industrial conditions.

(d) Syphilis of the spine is either much

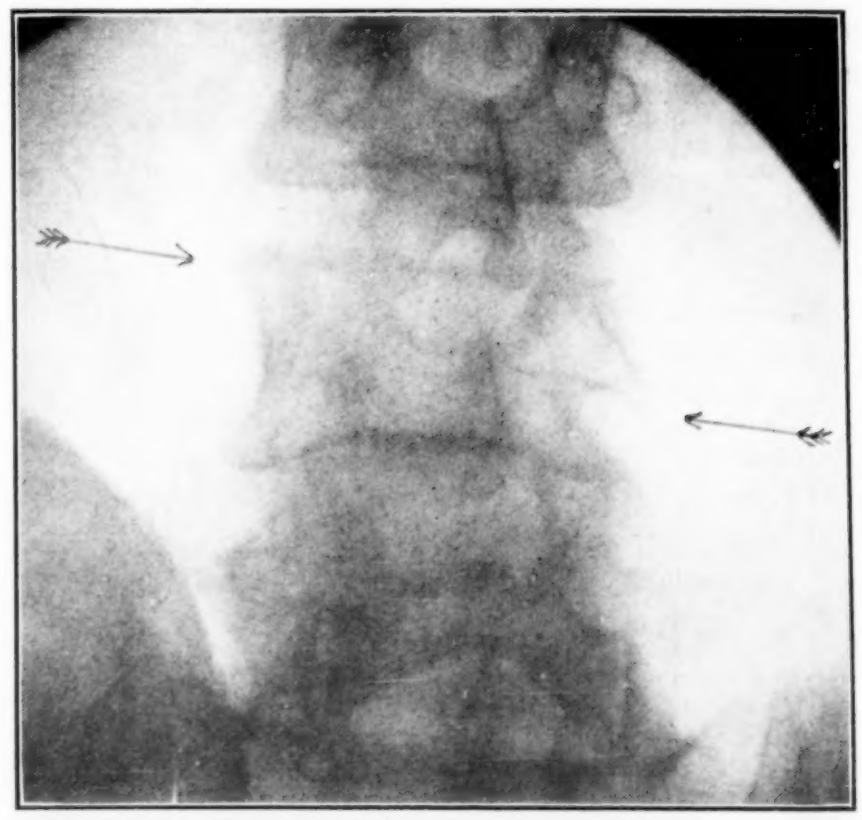

FIG. 10.—Hyperthrophic arthritis of the spine. Note the marked speculation on the right of the picture and the early changes on the left.

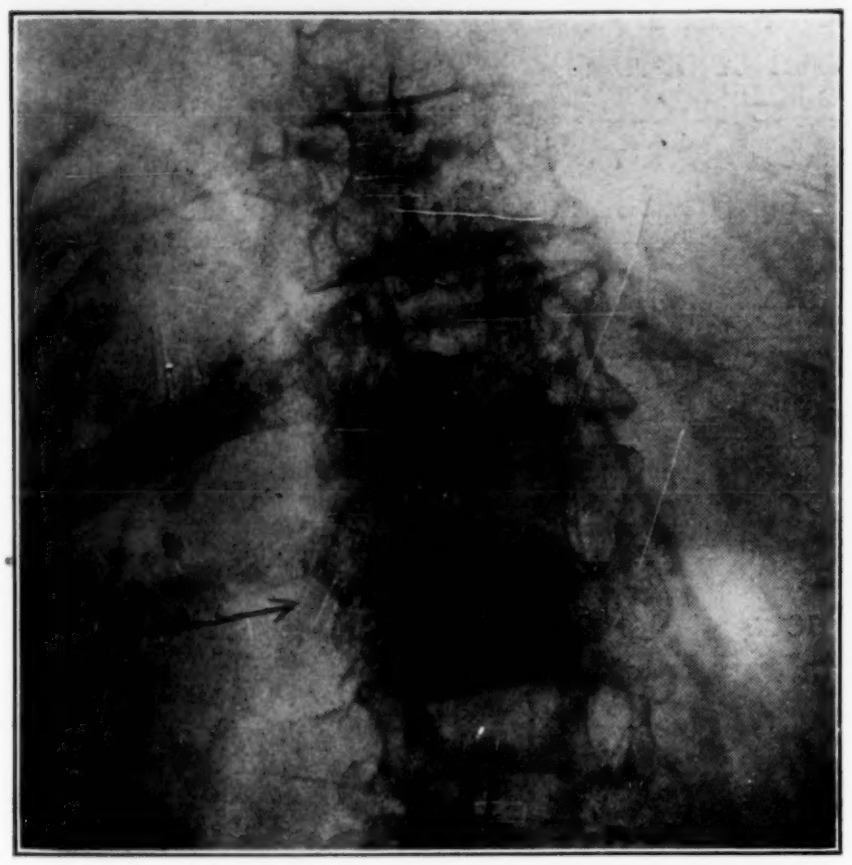

FIG. 11.—Extreme structural atrophy of vertebræ in connection with tuberculosis of the spine. Lesion between the 1st and 2d lumbar vertebræ.

less commonly recognized in certain sections of the country than in others, or is actually in these sections less common. It is a frequent diagnosis on the Pacific Coast and a rare one on the Atlantic. Congenital syphilis, as well as acquired, is difficult without the Wasserman test to differentiate from tuberculosis, but its response to treatment, of course, is quite different. Tabetic lesions of the vertebrae have long been recognized. They are not common, but they should not be forgotten.

(e) Osteomyelitis (Fig. 13) is usually acute in its onset, and its outcome depends upon whether the bodies or the articular processes and the laminae are the seat of the infection. If the disease is in the bodies, the abscess frequently ruptures anteriorly, infects the viscera and is fatal. In the processes or laminae the disease may be drained posteriorly.

(f) Neoplasms (Fig. 14) must be mentioned in connection with back strain, and especially with relation to sciatic pain. Bilateral sciatic pain is rare in static and traumatic strain. It is common in spinal cord tumors and malignant disease of the bones. Epithelioma of the bones always represents a metastasis. The extreme punctate atrophy of a chronic tuberculosis may be mistaken for myelomata. Small, round-celled sarcoma, we have personally seen very rarely, if ever, in the spine. We have seen occasionally a giant cell sarcoma or as it has been recently called, hemorrhagic osteomyelitis.

(g) Under this heading of disease we must mention the group of so-called functional or hysterical backs. In the absence of fairly definite stigmata this diagnosis should rarely be made. Railroad spines may have occurred as a result of railroad accidents, but the name is not descriptive of a typical picture. The diagnosis of functional spine is almost as rarely justifiable as neurasthenia.

Summary

To answer the question of the title "Back Strain—An Accident or a Disease?" we may say either or neither or both. Having been paid our patients' or our companies' money, we must make our choice. The choice may today be based on knowledge and not on surmise. We must approach the choice without preconceived notions. We must refuse to consider our duty done when we make a diagnosis of lumbago or sciatica or back strain, or I regret to say, nowadays, of sacro-iliac disease or railroad spine.

Unless we make the most careful examination and unless we employ the most accurate diagnostic methods we shall continue to be puzzled by the persistence of the symptoms and our patients will continue to seek and often obtain osteopathic, chiropractic and nostrum relief. We must know why and when and how to

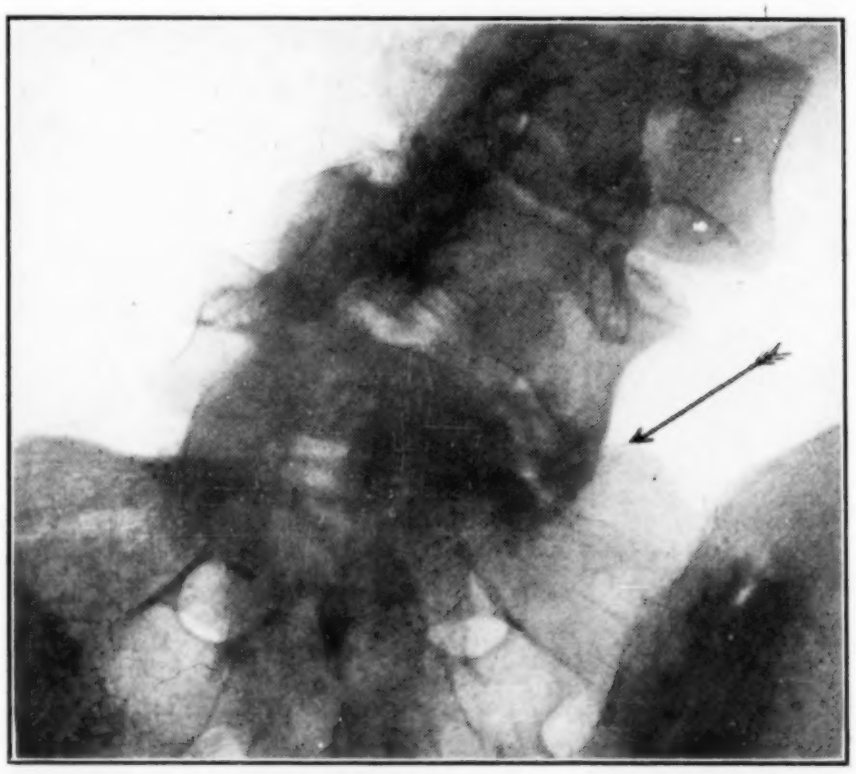

FIG. 12.—Old asymmetrical disease, probably healed tuberculosis, between 4th and 5th lumbar vertebræ. Note marked scoliosis as a result.

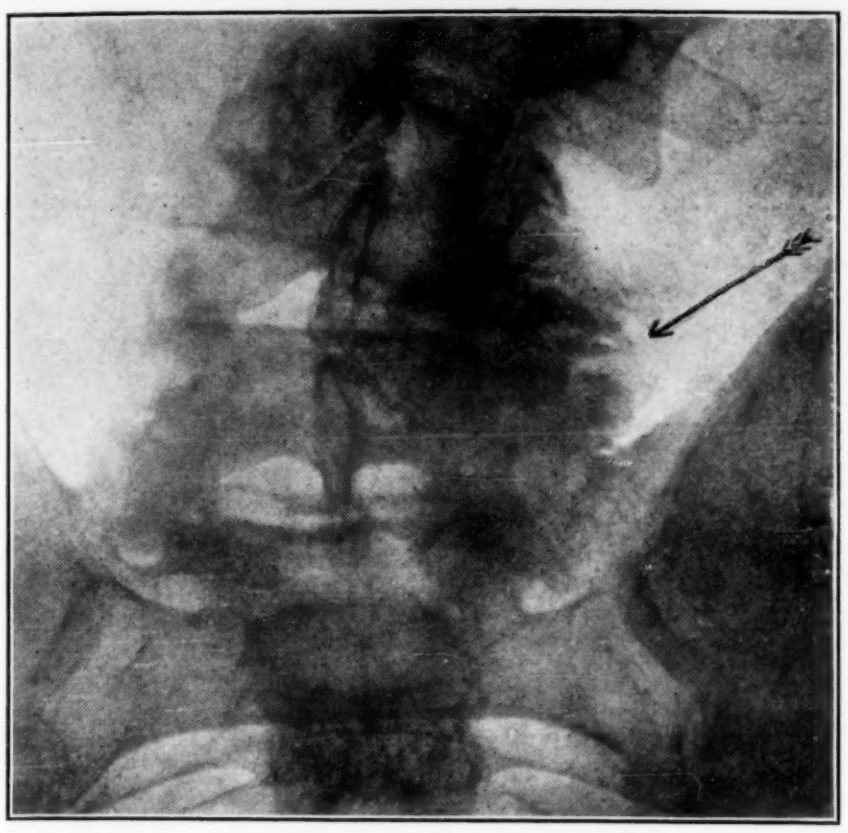

FIG. 13.—The osteomyelitis or Charcot vertebræ. Possibly an old fracture.

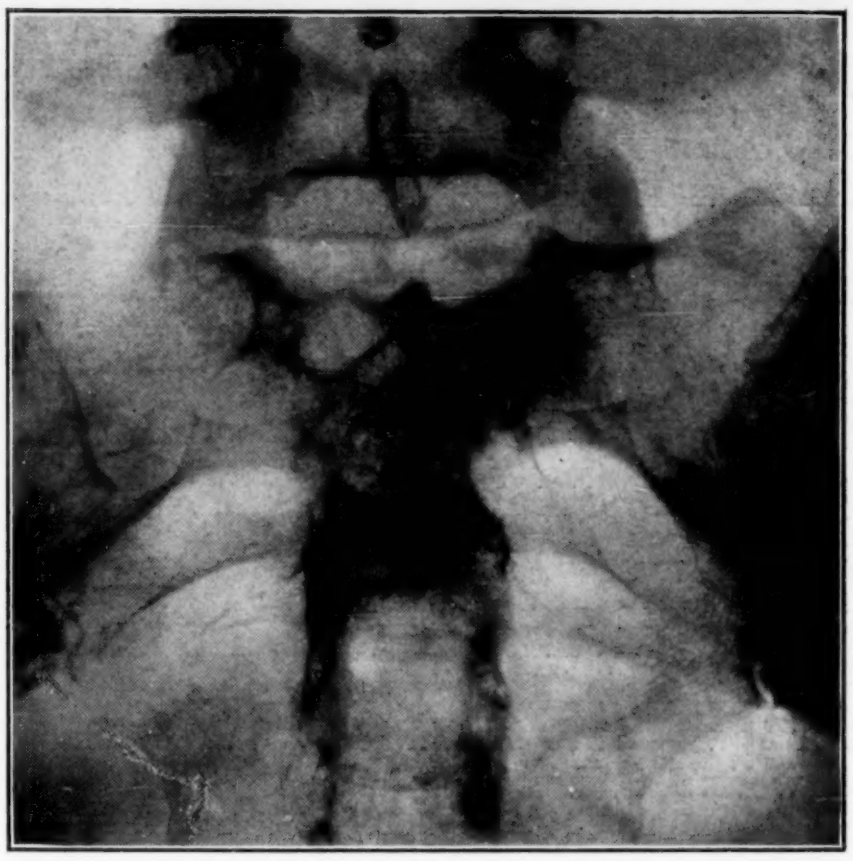

FIG. 14.—Diffuse epithelioma of the ilia and sacrum from metastases. (Morrison.)

protect, and why and when and how to exercise. Forcible manipulation is not without danger nor is a back brace a panacea.

I trust, gentlemen, I have left the sense of confusion in your minds. Until we sense this confusion we cannot begin to be clear-headed. But there must be a way out of our confusion and it is necessary that we find it. The considerations we have merely suggested seem to us the necessary first steps toward our emergence.

I want to leave with you this: That the diagnosis of hysterical spine should rarely be made; that the diagnosis of railroad spine means nothing; that the diagnosis of functional spine is usually a confession of our ignorance of the true cause of the trouble. My experience in medico-legal cases is not extensive, but it is large enough to have made me blush for the surgeon who contents himself with the diagnosis of sciatica or lumbago or functional spine, the nature of which neither the patient, the jury, nor the surgeon himself can understand. On the other hand, the plaintiff's lawyer or physician may demonstrate clearly by X-rays the separation of the sacro-iliac joints or the displacement, and secure his verdict by proving to the uninitiated jury his contention. *We* may know that the separation he points out is entirely within normal limits. *We* may know that it has probably nothing to do with the symptoms. *We* may know that the displacement he demonstrates is due to the asymmetry of the X-ray tube and not to the asymmetry of the patient, but *we* should be the first to demonstrate these facts. Only the careful analysis of each case and the knowledge of the normal and the commonly symptomless variations will allow us to do so. We shall advance neither the interest of science nor of our patients nor of the companies we represent until we take time to analyze thus our cases of back strain.

I wish to express my thanks to Drs. Morrison and George of Boston, for several of the X-ray plates and lantern slides.

IS WAR TIME SURGERY APPLICABLE TO INDUSTRIAL SURGERY? *

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WAR surgery in the Evacuation Hospital zone was essentially the surgery of extensive lacerated wounds and compound fractures, this group comprising about 90 per cent. of all the lesions produced by shell, machine gun and rifle fire. The main impression that I received of high explosive wounds was their multiplicity and their extent, for it was rare to find only one wound or a wound that failed to look as if a dissection had been attained, many of them producing an exposure as if in a biopsy.

The immediate complications of war wounds were shock and infection. The shock often was due to blood-loss in combination with cold, exposure, hunger, thirst and exhaustion. The infection was frequently of the gas bacillus or other anerobic type, although for the most part the ordinary pus producing staphylococci and streptococci prevailed. We actually dreaded the streptococcus more than the gas bacillus for it was more common, more resistant and persistent, practically always present in bone infections. The lesions of the organs within the skull, thorax and abdomen were fairly numerous and they also were characterized by their multiplicity and severity. Of all war injuries, I believe those of the skull and abdomen more closely paralleled civil injuries than any others because only those relatively minor survived long enough to reach us in our forward zone. Chest wounds were unique and of a type almost never seen in civil practice and much to our surprise and satisfaction many of them did surprisingly well when subjected to the special technic devised for their relief. Joint wounds were also treated after a new method and they likewise responded well. Purposely I have said nothing as to vascular or neural injuries, for their treatment is quite similar to the less grave injuries of civil life.

The adaptation of war surgery lesions to those of industrial surgery finds no *exact* parallel because of the marked difference in their causation, the inherent differences in the types of infection and the all important element of the time elapsing between the onset of injury and the receipt of the patient. One other salient factor is the age and physique incidence of the soldier patient as compared with the patient in civil life. This is a cardinal difference to which must be added the morale of the soldier, the will to get well, an attribute all important in the outcome. Our patients in civil life are often beyond the draft age, their physique is often below par, their vitality often has been affected by disease, malnutrition or by alcohol. The soldier patient has an asset in his youthful, sturdy physique; the civil patient often has a pathological deficit to which the injury adds another burden.

This brief review prepares the way to answer in the affirmative the inquiry propounded by the title of this paper. War surgery is applicable to industrial surgery in the following particulars:

I. *Wounds.* Experience taught us that infection was the only real foe to primary union and that if we could rid the wound of devitalized tissue, healing would proceed favorably because germs could not live in healthy surroundings. This lead to what is termed "mechanical sterilization," as distinguished from "chemical sterilization." The former means that with the knife and scissors all damaged tissue is removed until the part looks healthy, until it bleeds and until muscle contracts. This process of cutting away is known as "Debridement" (literally unbridling) and only such parts are removed as are manifestly unhealthy. It cannot be stated too strongly that this form of sterilization is not sacrificial and that the process is more like paring the surface than exsection of it. In cases thus treated within the first

*Read before the Fourth Annual Convention of the American Association of Industrial Physicians and Surgeons, Atlantic City, June 9, 1919. Received for publication June 16, 1919.

18 hours, experience showed that primary suture could be made with safety and success. Cases arriving after the 18-hour period were treated in the same way, but the suturing was postponed until between the third and sixth day, bacteriologic tests meanwhile proving the presence or absence of streptococci. If these germs were absent even if others were present, this "delayed primary suture" method (or "primo-secondary" suture) would prove successful if the wound appeared clinically healthy.

Wounds received late and already suppurating must be subjected to the procedure known as "chemical sterilization," and when relatively pus free they too can be sutured, the method being known as "secondary suture." Mechanical sterilization may be termed the aseptic method; chemical sterilization the antiseptic method. Antiseptics of many kinds were employed either in the form of the familiar wet dressings or by intermittent instillation after the method of Carrel, using the hypochlorite solution of Dakin made after Daufresne's formula.

The application of these methods to civil surgery is very easy and is called for principally in lacerated wounds associated with bruising or fraying of the soft parts. The vast majority of industrial accidents are cared for promptly and the types of infection are relatively less virulent; therefore the essential element in success is to rid the wound of contained infection by debridement or antiseptics, bearing in mind the possibility of re-infection unless our procedures are carried out with every aseptic detail.

I would emphasize the need for exploring the entire wound under the guidance of the eye, clearing out of it all foreign material, stopping hemorrhage, and then flooding it with full strength iodine before suturing. This applies to the ordinary wound in which the edges do not require paring because they are neither bruised nor frayed. I believe that every wound so sutured should be drained by a strand of silkworm gut or rubber, the drain to be removed in 24-48 hours. If there is any question as to the sterility of the wound at that time, the secretion at the inner end of the drain can be examined and the exact nature of the organisms definitely determined. For wounds already infected, gaping or with open surfaces, there is no better treatment than the Carrel-Dakin procedure. This unfortunately requires special preparation of the solution and a special technic for the application of it. The proper carrying out of this technic is in effect an aseptic operation, it requires training, it is time consuming and it therefore cannot have wide application. Aside from the value of the solution, I believe that one of the great elements in the success of the method is the care with which it has to be applied and the improbability of re-infecting a wound by contact with soiled instruments or fingers. Failure to sterilize an ordinary wound by this treatment is to be charged more to the surgeon than to the method. To use the Carrel-Dakin ritual is not the road to wound salvation unless all the precepts are carefully followed. In this connection it is profitable to state an item of war experience to the effect that persistent streptococci infection usually means the presence of infected bone; and that persisting discharge usually means the presence of a foreign body, often frayed or otherwise devitalized soft parts.

II. *Fractures* should be much better treated in future as a result of war experience, not only because a compound fracture can be converted into a simple fracture by primary or delayed primary suture, but also because the profession in general have become aroused to the importance and technical difficulty of this branch of surgery. Industrial surgeons have long recognized the need for better care of this class of case even as they have realized that the treatment requires more skill and judgment than is ordinarily needed to remove an interval appendix or to perform a herniotomy. The various splints used for war fractures of the long bones should have a wide application. First-aid men should and can be taught to apply Thomas splints for the arm or leg and these same appliances with appropriate overhead frames can be used for the treatment of nearly all the fractures of the extremities. In the treatment of broken bones it is important to recognize that there are two grades or classes, speaking generally. One class is what may be termed the *displaced variety*, the other the *non-displaced variety*. The sec-

ond class requires little if any attempts at reduction or setting and our only concern is to keep the part at rest by suitable splintage. The other class, however, requires setting of the displaced fragments before we can expect a splint to be of service. In war experience practically all the cases were of this displaced type, badly compounded and comminuted for the most part. Many limbs were saved by rigidly applying mechanical sterilization technic to the soft parts, removing bone fragments that were devitalized or detached. This same principle is applicable to civil surgery and no limb should be amputated unless the main vascular supply has been severed, the main bone is hopelessly pulpified, or infection is of such a grade that systemic symptoms are present.

Fracture of the femur was the greatest fracture problem of the war and the majority of surgeons in fracture centres came to rely more and more upon traction applied to the bone itself rather than when applied to the soft parts overlying the bone. The English surgeons advocated the calipers traction device (known to us as the "icetongs") and I saw this in use in some of the British hospitals during a visit to England and Scotland after the armistice was declared. This is a simpler method than the Steinmann nail and the application can be made under local anesthesia.

The Finiochetto stirrup is used by some French surgeons for the treatment of all fractures of the lower extremity, as this device when placed over the concavity of the os calcis produces a very considerable traction on the whole lower extremity. The Chutro or Hawley modification makes the introduction rather easier and more exact. The calipers and stirrup will be for me standard methods of treatment in lower extremity fractures to the exclusion of all other means in selected cases.

Too much stress cannot be placed on the fact that in a fracture the broken bone is only one part of the damage inflicted, and that the associated injuries to the soft parts and joints are often more important. This means that after a broken bone is suitably reduced and splinted, our main treatment should be directed toward the associated lesions in the contiguous parts. The day has gone by when we can discharge our patient as cured when union has been attained. I believe we do most for the patient when we place the fractured bone in a splint that gives access to the limb for massage and motion designed to prevent atrophy, and the adhesions growing out of teno-synovitis, synovitis and arthritis. There are few fractures once set in which massage and motion are contra-indicated from the very beginning of treatment. There are "three R's" in fracture treatment, namely, Reduction, Retention and Restoration, and of these three the last named is really the most important and yet the most neglected. The restorative, re-educative or reconstructive side of war surgery should and can be carried on into civil surgery, but the prevention of much of the crippling from fractures can only be attained by recognizing that while reduction and retention are necessary preliminaries, restoration of function should begin early and not after the fracture has healed. Let us not forget also that deformity of bone does not necessarily mean diminution of function. Witness the almost constant deformity following fracture of the clavicle with perfect function of the shoulder if we do not immobilize the joint until we virtually seal it by too long fixation.

III. *Joint Injuries* in war taught us many valuable clinical facts to which Willems of Belgium, in particular, called our attention. It has now been demonstrated that the synovial lining of a joint is capable of standing a relatively large amount of trauma and in that respect as in others it resembles the peritoneum.

Joint penetration as from recent wounds or foreign bodies is now treated in precisely the same way as wounds of the soft parts, that is by the mechanical sterilization which cuts away sparingly the tract leading to the joint so that nothing but a healthy surface is left. Ether is then used to wash out the joint cavity, all bleeding is stopped and the synovial membrane is separately sewed, then the muscle-fascial layer alone, and finally the skin. No drainage whatever is used. Then instead of putting the joint at rest in a splint, only a soft dressing is applied and the patient is made to move the joint each day through increasingly wide angles. In a joint already the site of sep-

tic arthritis, a lateral or bilateral incision is made giving access to the joint interior, it is washed out with ether and again no drainage is inserted, but instead the patient is forced to move the articulation so that the normal flexion and extension literally squirts the pus out of the joint cavity. In a septic knee, the patient is made to walk; in an elbow, exercises are prescribed enforcing bending and straightening the arm and forearm. No splintage of any kind is used and motion is the key note of success as opposed to rest. This type of treatment has an application in many civil injuries and it is based on enough experience to warrant advocacy. Ordinary synovitis of the knee will in future be treated by me by lateral aspiration until all the fluid is removed and then the patient will be made to walk. Re-effusion will be treated by aspiration as often as may be necessary and in this way the disability period should be much lessened and I feel sure there will be far less stiffness from prolonged immobilization. In civil life this procedure of immediate mobilization was long ago successfully used in the treatment of a sprained ankle and the principle has now a very much wider application.

IV. *Blood Vessel Injuries* in some cases were successfully treated by placing over the puncture a flap of fascia and this procedure may have a limited application in civil life. Some French surgeons have reported a few successful cases in which an elbowed silver or glass tube has been placed in the lumen of a main vessel to bridge a gap until collateral circulation has been restored.

V. *Nerve Injuries* were exceedingly numerous but in general the outcome of suture has been disappointing inasmuch as a very long period ensues before restoration occurs. The best practice seems to be to have the separated ends free of any fibrous tissue, to join them by numerous fine silk threads and not to wrap the place of junction in fascia or any other material. The application of this newer type of neurorrhaphy to civil life is of course quite evident. When a gap exists, the external cutaneous nerve of the subject is usually chosen to bridge the defect. In the musculo-spiral and in the peroneal types of involvement, transplantation of tendons at the respective joints is a favorite method as this can be done at once so that the wrist drop and ankle drop may be overcome more promptly than if neurorrhaphy alone was done.

VI. *Visceral Injury.* In the *skull,* foreign bodies are brought into better view if a soft rubber catheter is introduced into the wound tract and through this large amounts of sterile water are poured and thus pulpified brain tissue is removed by hydraulic pressure. A number of these cases have been operated upon under local anesthesia and experience seems to indicate that primary closure of the dura and skull should be made, when possible.

I know of nothing new in connection with the treatment of fractured skull, except that in certain cases with mounting blood pressure, relief is sometimes obtained by repeated spinal taps. The same applies to the delirium seen with head injury and to the headache so often complained of. For hernia cerebri, spinal tapping should be energetically tried before operation is attempted.

In *thoracic surgery* a new chapter has been written and this cavity is no longer a zone to be approached with temerity. Fortunately the war types of injury are rarely duplicated in civil life. We no longer need special pressure apparatus before opening the chest cavity and the collapsed lung can be brought out through the opening between the ribs and handled like a loop of intestine. The parallel is so apt that one French surgeon refers to the procedure as "laparotomizing" the chest. In empyema we have learned that repeated aspirations, with or without the injections of ether, will often effect cure instead of rib resection. We also know that early operation is not advisable in certain cases and that the main operative indication is the *pressure* from and not the *presence* of the effusion. After rib resection we treat the cavity on the theory that chemical sterilization will diminish the secretion and when this is accomplished and no streptococci are present, we close the wound by secondary suture and cut short what is ordinarily a very prolonged convalescence.

In *abdominal surgery* no particular changes are to be noted. The cases of abdominal war wounds that reached the hospitals were unfortunately often dying from shock, hemorrhage or peritonitis.

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Plastic surgery has had ample scope and many of the deformities from war wounds are of course more or less equalled in civil life and can be subjected to the same kind of advanced treatment of which no special mention need be made.

Burns of the primary degrees are benefited by the various paraffine dressings with which you are all familiar and of which I need say nothing further.

The main lessons of war surgery to be carried over into industrial surgery are connected with

a. Wounds of the soft parts.
b. Fractures.
c. Joint injuries.
d. Thoracic injuries.

a. In recent *wounds*, we can improve our results by mechanical sterilization in all associated with bruising, fraying or devitalization of the edges. In those already infected, intensive chemical sterilization will do much to reduce infection leading to the stage where secondary closure can be made, thus diminishing the period of prolonged cicatrization.

b. In *compound fractures* we can treat the wound by mechanical sterilization, suture it promptly and convert the case into a simple fracture. *Simple fractures* of the long bones are effectively treated by the army type of splints, that is by the Thomas or Jones variety of splints. For femur fractures, the calipers will be found efficient. For some thigh and many leg fractures, the stirrup will give excellent results. We must not forget to regard a fracture as essentially a lacerated wound of bone and with that conception of the lesion seek to coapt the fragments so that primary and not secondary union may be attained. To carry out the analogy the splint can be regarded as a suture, and like the latter, is to be removed when it has served its purpose. Regarding a broken bone in this fashion will lead us to a more rational form of treatment so that our attention will be directed quite as much to restoration of function as to attainment of union. If we will look upon traumatic osteomyelitis not as a special disease but only as an infected wound of bone we will alter our treatment so that we will proceed to sterilize chemically the infected area, and when this is accomplished, we will remove the slough or scar in the bone, fill in the cavity by muscle or fascia or both, and then close by secondary suture. It will be quite useless to try to sterilize an infected area of bone unless adequate exposure is afforded, and hence in many cases liberal incision and drainage will be the first object of our attack. This conception of osteomyelitis means that we will no longer wait for a sequestrum (literally a bone slough) to separate spontaneously, but we will by adequate exposure open the area for the contact of our antiseptic. Dakin's solution acts especially well in bone infection, applied by the Carrel technic or by wet gauze tapes snugly placed into the recesses of the cavity, replacing them each 24-48 hours. This last method was first called to my attention by my friend, Lt. Col. George Hawley, who has had much experience with it.

c. Joint injuries in future will be better treated by early mobilization applying the principles of mechanical and chemical sterilization. Rigid and prolonged splintage in joint injuries is no longer to be advised, and the ordinary accident of industry affecting an articulation will profit by this new form of management.

d. Thoracic injuries will profit also, notably in the treatment of empyema and the removal of foreign bodies from the chest cavity. It appears to have been conclusively demonstrated that of all the tissues the lung has the greatest resistance to ordinary infection, doubtless because of the vascularity and oxygenation, and for this reason early attack may be postponed with greater safety than in any other cavity of the body.

Very little that is absolutely new has come out of the war experience, inasmuch as the principles of surgery are well standardized; the wider application of these principles under diverse and adverse conditions has, however, focused our attention on many problems that are met with in civil traumatic surgery. It is interesting to note that the same methods of wound treatment used in this war were also recommended as long ago as the Napoleonic wars by the famous French surgeon, Larrey. Indeed, Louvard (with whom I was associated for a time in France) has shown in his recent article in *Presse médicale* that many modern ideas revert to the time of Hippocrates.

BOOK REVIEWS

Vital Statistics: An Introduction to the Science of Demography. By George Chandler Whipple, Professor of Sanitary Engineering in Harvard University, Member of the Public Health Council, Massachusetts State Department of Health. First edition. Pp. 517, with illustrations. New York: John Wiley & Sons, 1919.

Vital statistics is the bookkeeping of humanity, but there are few good bookkeepers. There are still fewer expert accountants who understand the mathematics and mechanics of statistics. The ability to use vital statistics in public health work is an important part of the training of the modern health officer. Professor Whipple has done a real service by writing a clear, concise book on vital statistics as an introduction to the science of demography. Broadly speaking, demography is the statistical study of human life. Students of every branch of hygiene and sanitation owe a debt to Professor Whipple for the plain, accurate and lucid treatment of the subject. Even those who have a dislike for arithmetic will find the subject made easy and convincing.

The book makes no claim to be an exhaustive treatise or a compendium of facts; it is merely a guide to the study of vital statistics, an introduction to the great world-wide science of demography—a science yet in the magmatic stage, not yet crystallized. The Great War is bound to develop this science, because hereafter all the nations of the earth must know each other better, and this knowledge, in order to be usable, must be condensed into statistical form.

Specifically the book tells what statistics are and what they are not; it shows how to express vital facts by figures, how to tabulate them, and how to display them by diagrams; it shows how to compute birth-rates and death-rates and how to analyze a death-rate; it shows how to adjust and standardize death-rates and how to make life tables; it emphasizes the need of using vital statistics with truth, with imagination and with power. For the convenience of school instruction, exercises and questions to incite further study are given in each chapter.

The chapters deal with: Statistical arithmetic; statistical graphics; enumeration and registration; population; general death-rates, birth-rates, marriage rates; specific death-rates; causes of death; analysis of death-rates; statistics of particular diseases; studies of deaths by age periods; probability; correlation; life tables; and a commencement chapter.

The method of preparing graphs will be found particularly useful for those who have the difficult task of interpreting vital statistics to lay and professional audiences. This section should be carefully studied by the makers of health reports and also by public health educators. There is no group of observers whose opportunities for collection of valuable statistical material equal that of the industrial physician and it is fair to say that the deficiencies in their training make them unequal to the proper handling of the facts in their possession. Realizing this truth, educators are including thorough courses in vital statistics both in the curricula for health officers and for industrial physicians. To the latter group this book of fundamental principles will be found extremely valuable.

The publishers deserve a special word of commendation for the sensible example of the bookmaker's art, for it is evident that they have given careful thought to the way in which they have made Professor Whipple's book on *Vital Statistics*. The convenient size, the flexible cover, as well as the print and paper deserve favorable commendation. The volume is well illustrated and has a good index.

The book is dedicated to "The Students of Vital Statistics in the School of Public Health of Harvard University and the Massachusetts Institute of Technology."

Vital Statistics is timely and will be useful to students and practitioners of all phases of the art of sanitation and the science of hygiene. It is indispensable for health officers and will be found useful for sanitary engineers, nurses, social service workers, and medical students.—*Milton J. Rosenau.*

How to Prevent Sickness. By G. L. Howe, Medical Director, Eastman Kodak Company. Pp. 203, with illustrations. New York: Harper & Brothers, 1918.

This is a well written and well illustrated book of popular type which should find a place in many factory libraries. The representations in regard to scientific medicine are clear and accurate.

The industrial physician desiring to formulate a campaign in health education in his plant will find in this manual a valuable supply of illustrations, tables, and text which it is a pleasure to recommend.—*Katherine R. Drinker.*

What We Eat and What Happens to It. The Results of the First Direct Method Ever Designed to Follow the Actual Digestion of Food in the Human Stomach. By Philip B. Hawk, Ph.D., Professor of Physiological Chemistry of the Jefferson Medical College of Philadelphia. Cloth. Pp. 232. New York: Harper & Brothers, 1919.

This is a collection of popular articles reprinted from magazines. They have a laboratory basis in very numerous observations made upon volunteers who submitted to the passage of the stomach tube for the withdrawal of test meals. The chief criterion of digestion is assumed to be time required for the disappearance of various foods from the stomach. This is rather an inadequate standard but it is still a matter of interest. The discussion is chatty and entertaining. The book contains few prohibitions and is generally cheerful and reassuring reading.—*Percy G. Stiles.*

BOOKS RECEIVED

Books received are acknowledged in this column, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Healthful Schools, How to Build, Equip, and Maintain Them. By May Ayres, Jesse F. Williams, A.B., M.D., Professor of Hygiene and Physical Education, University of Cincinnati, and Thomas D. Wood, A.M., M.D., Professor of Physical Education, Teachers College, Columbia University. Cloth. Pp. 292, with illustrations. Boston: Houghton Mifflin Company, 1918.

Effects of the War upon Insurance, with Special Reference to the Substitution of Insurance for Pensions. By William F. Gephart, Professor of Economics, Washington University, St. Louis. Carnegie Endowment for International Peace. Preliminary Economic Studies of the War, No. 6, edited by David Kinley, Professor of Political Economy, University of Illinois. Member of Committee of Research of the Endowment. Paper. Pp. 302. New York: Oxford University Press, 1918.

Effects of the Great War upon Agriculture in the United States and Great Britain. By Benjamin H. Hibbard, Professor of Agricultural Economics, University of Wisconsin. Carnegie Endowment for International Peace. Preliminary Economic Studies of the War, No. 11, edited by David Kinley, Professor of Political Economy, University of Illinois, Member of Committee of Research of the Endowment. Paper. Pp. 232. New York: Oxford University Press, 1919.